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SIGNS AND SYMPTOMS OF THE ACUTE STAGE OF ANTERIOR POLIOMYELITIS IN THE 1931-1932 EPIDEMIC.[1]

By S. F. McDONALD, M.D., M.R.C.P.,

Honorary Physician, Hospital for Sick Children, Brisbane.

I TAKE it that the essential feature of this paper is to be a description of the clinical manifestations of poliomyelitis as I saw them during the recent epidemic. In other words, I have, to quote Sigmund Freud, shut myself up with the facts until the facts should speak to me. Thus, except for acknowledgements to the work of such people as Draper[4] in America, and above all to Dr. Jean Macnamara and her fellow workers in Victoria, I shall not quote from authorities, but from our own cases. For those who desire an excellent clinical account of the disease I would suggest Dr. Macnamara's articles in THE MEDICAL JOURNAL OF AUSTRALIA,[1] [2] [3] or that storehouse of clinical pædiatrics, John Thompson's "Examination and Treatment of Sick Children".

The recent epidemic differed from previous epidemics in that for the first time the medical profession (and later on the public) were seized of the importance of the preparalytic stage.

As one of those responsible for much of the newspaper propaganda which was published at the beginning of the epidemic, I stand absolutely impenitent for the course we took. We shall no doubt hear much talk tonight reflecting on scare tactics and creating a panic. Granted that in many cases medical men, of whom I was one myself, were called out quite unnecessarily, the fact remains that many cases were recognized early owing to the parents having read our accounts in the Press.

This paper is based on records of patients seen in the Hospital for Sick Children, of several seen at

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932.

private practice, and, by the courtesy of Dr. Pye, Assistant Medical Superintendent, of nine patients treated at the Brisbane Hospital.

I have excluded patients who were admitted to hospital weeks after the onset, when no accurate account could be obtained of the acute stage; and I have included very few whom I did not actually see myself.

The cases may be divided roughly into the abortive and paralytic. The latter were preceded by a pre-paralytic stage indistinguishable from the abortive type, but progressing to paralysis, unless checked either by the patient's resistance or by the administration of serum.

Shortly, in the typical "dromedary" curve (Chart I), originally described by Draper,[5] there is an initial stage lasting from two to six days. There

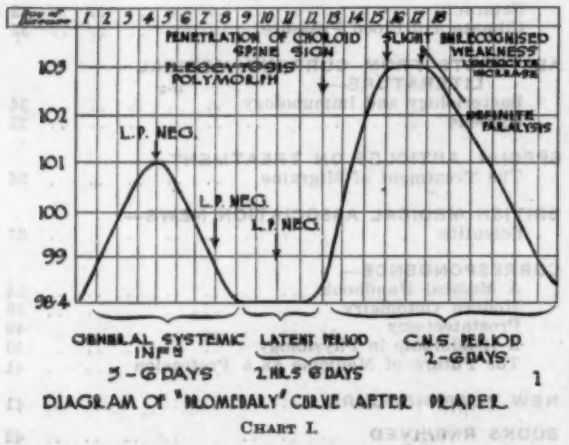


CHART I.

are all the manifestations of any acute feverish illness: headache, fever, vomiting, lack of appetite, and above all, a dengue-like state. At this time there is usually neither stiff neck nor spinal rigidity, signs which Draper considers are the evidence of the penetration of the chorioid plexus and the infection of subarachnoid spaces. There is, however, the intense resistance to anything in the way of voluntary movement or outside disturbance, so characteristic of dengue fever. In many children, the condition goes no further; we have an abortive attack and the child recovers. (We saw several such cases. One occurred early in the epidemic; we did not recognize it at the time, but possibly it was a source of infection to two others.)

In others, after an interval, which may be only a few hours, the temperature again rises; headache, nuchal rigidity and spinal rigidity become definite; and unless the patient's resistance is sufficient or serum is administered, paralysis occurs. (Chart II.)

It is possible that much of the foregoing will require modification in view of the work of Hurst and Fairbrother, which Dr. Duhig will put before you presently.

Very few cases showed this regular sequence; the preparalytic stage varied from a few hours—in two cases it was apparently non-existent—to twenty-three days.

Draper points out that the cells found in the spinal fluid change, too, with the stage of the disease. In the early stage (the first hump) they are usually absent, with the commencement of the second hump the number is not very high, and they are polymorphonuclear in type. With the change of the stage from general to local invasion, the lymphocytes and mononuclear cells increase in numbers, so that Dr. Macnamara considers this increase as a danger signal calling for increased doses of serum.

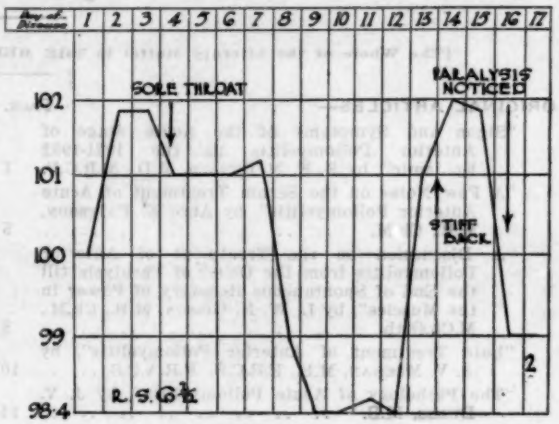


CHART II.

In only one case could we be certain that the preparalytic stage was absent.

In this the child, a baby aged two years, the daughter of a medical man who had had wide experience of the disease, was quite well until she woke from a normal sleep at 2 p.m. When seen at 2.30 p.m. the child was feverish, irritable, and unwilling or unable to walk on the right leg. Neck and spinal rigidity were present and it was decided to perform a lumbar puncture. The fluid was slightly blood stained, but contained an excessive number of white cells, about 14 per cubic millimetre. Serum was given, 20 cubic centimetres intrathecally and 20 cubic centimetres intravenously, and the child was sent into the Hospital for Sick Children. There weakness of gluteal muscles was obvious and the child was put into plaster. She made a complete recovery.

Another, also a child of two years, (according to the parents) had no previous symptoms whatever.

The duration of the preparalytic stage was variable.

One child, a patient in hospital, puzzled us thoroughly. (Chart III.) He was admitted originally for fever of uncertain origin at the very beginning of the epidemic, and some time after admission an acute *otitis media* was found in one ear. This was incised with only transitory benefit, as he continued an irregular temperature with stiff neck and back for nineteen days. On the tenth day lumbar puncture was done; we withdrew 30 cubic centimetres of almost clear fluid under increased pressure, with a cell count of 9 per cubic millimetre and no increase in globulin. Finally his right deltoid became paralysed; the temperature fell; and from being morose and irritable he became a bright and lovable child.

The dromedary type of temperature curve is in effect very like the temperature curve seen in dengue fever, and it is certain that had we had a dengue epidemic at the same time, the difficulties of diagnosis would have been vastly increased. As it was, the presence of an obscure febrile illness much resembling dengue fever, but without the relapse and without the rash, often made diagnosis difficult. (Chart II.)

Sunstroke also might have complicated the diagnosis; it is almost unknown here.

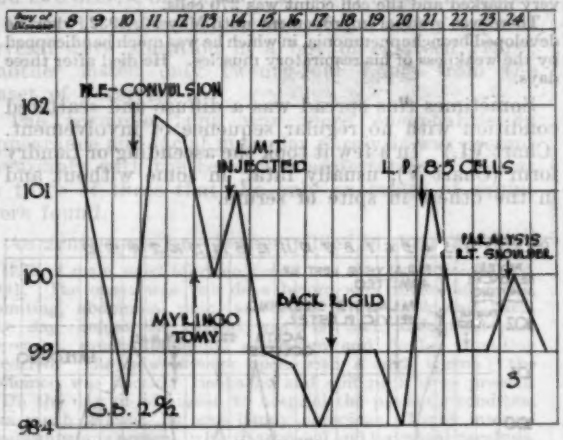


CHART III.

In this country the difficulty of diagnosis from tuberculous meningitis has not the same force as in Victoria.

One child, however, whom I saw in consultation with Dr. Davison at Newmarket, was, I was convinced at the time, suffering from tuberculous meningitis. It was only when the complete fluid examination was done and paralysis appeared two days later that we were undeceived.

Influenzal meningitis is sometimes a difficulty. At the Hospital for Sick Children we recently had a case of fatal influenzal meningitis, with a spinal cell count of only 180.

The accompanying table shows the various signs and symptoms and their relative frequency.

TABLE I.

Showing Summary of Signs and Symptoms of Acute Stage in 128 Cases in 63 Males and 65 Females.

Sign or Symptom.	Present.	Absent.	Not Noted in Records.
Fever	126	2	
Headache	119	7	2
Backache	117	9	2
Dengue state	119	9	0
Delirium	16	112	
Vomiting	68	43	17
Constipation	51	74	3
Diarrhœa	8	116	4
Retention of urine	11	23	94
Squint	3	106	19
Dry tongue	68	60	0
Nuchal rigidity	98	22	8
Spinal rigidity	106	15	7
Amoss's sign	48	40	40
Muscle tenderness	83	34	11
Deep reflexes	67	44	17
Babinski's sign	3	111	14
Kernig's sign	20	86	14
Increased spinal pressure[1]	84	20	24
Increased cell count[2]—			
0-10	26		
10-50	30		
51-100	14		
101-200	19		
Above 200	5		
Globulin increased	72	14	42
Majority of cells—			
Polymorphonuclear cells	33		
Lymphocytes	14		
Neutrophile cells	77		
Lymphocytes and polymorphonuclear cells	4		
Paralysis	69	59	

[1] In 33 cases lumbar puncture was not done owing either to extreme illness of child or to paralysis being already present.

[2] In seven paralysed patients the count was under 10.

TABLE II.

Showing Distribution according to Age and Paralysis.

Age Groups	Number Affected.	Persisting Paralysis.	
		Number.	Percentage.
1- 2	12	2	16
2- 4	30	7	23
4- 6	33	4	12
6-10	26	5	19
10-15	17	5	29
15-20	7	3	
20+	2		

Squint was not present in any case, except during the paralytic stage in two cases, both exceedingly severe infections.

Tenderness of the muscles was common after paralysis had appeared, not before. Actual delirium occurred during the preparalytic stage in fifteen cases, it was more common in the paralytic stage.

The most constant signs were neck rigidity and spinal rigidity. Amoss's sign was always of doubtful value—if it were present spinal rigidity was marked. Nuchal rigidity and spinal rigidity required much care to elicit, to push the head of a sick child suddenly forward will in many cases provoke resistance, and it was essential that the test should if possible be carried out by the child, hence in older children the head between legs, and in smaller children, knee-kissing were the methods used.[1]

The spinal and nuchal rigidity is probably a protective reflex to guard the irritated posterior roots; it is comparable to the rigid right lower rectus of appendicitis. Tremor, upon which many writers lay great stress, we did not find a prominent symptom. There is little mention of it in the reports. Personally I look on it as simply part of the dengue state, where shakiness of every action adds to the general misery.

Roughly, we found the cell count proportional to the severity of the infection, thus a patient with a severe ascending infection developed a count of 630 cells, another of almost complete paralysis 240, though moderately high counts, for example, 140 and 170, were not incompatible with complete recovery, nor very low counts with paralysis. These points will be elaborated by Dr. Duhig.

Dr. Masel, of Stanthorpe, recently had under his care a patient with a cell count of 1,200—entirely lymphocytic. The child made a complete recovery after the administration of serum, notwithstanding an intercurrent attack of lobar pneumonia.

It may be said that we considered lumbar puncture justifiable in any child with headache, fever, prostration, stiff neck and back. In some cases all these signs were not present and we made a puncture on suspicion.

Lumbar puncture was always done under a general anæsthetic, although the pain of puncture might not be intolerable even in a child, yet to bend the back sufficiently would in most cases have been to inflict great suffering. Unfortunately the use of an anæsthetic precluded any exact measurement of the spinal pressure. Lumbar puncture was not done in 33 cases when the child was desperately ill, especially before the arrival of serum; nor later

[1] Photographs were shown illustrating spinal stiffness, nuchal rigidity and Amoss's sign.

in the epidemic, once paralysis had set in, since we considered in these cases it served no useful purpose unless we gave serum (which we could ill spare), was unnecessary for diagnosis, and increased the risk of spread. This view, Dr. Paterson will tell you, we were to modify later.

The paralytic stage was ushered in by a rise of temperature (often the peak of the second hump), pain and tenderness in the muscles, and then paralysis. (Charts I and II.)

Sex incidence was practically equal. Age incidence is shown; the greatest susceptibility appears to be in the age groups two to four and four to six. But examination of the children still in hospital with persisting paralyses shows the very interesting fact that the older children tend to be more seriously crippled. This I think bears out the view Dr. Meehan has expressed frequently that the paralysis is apt to be more severe in the more hard worked muscles. Thus in a larger child or young adult the lower limbs have to carry a considerable amount of weight and are damaged in proportion.

In a few cases there seemed to be sensory changes, but these children were very ill and it was difficult to be certain. Possibly it was due to disturbance in the posterior roots or, in the case of one child, where loss of pain sensation was present, to involvement of fibres crossing over in the grey matter.

In most cases the paralysis, having appeared, progressed no further, the temperature fell and the child was at once more comfortable, except for residual tenderness in the muscles. Such cases are here shown. The muscle tenderness was usually relieved at once by adequate splinting. Sometimes the paralysis became apparent only when the child was allowed up. On the contrary in other cases, the paralysis progressed; and the severe generalized form was seen. (Charts IV, V, VI.) (In five the

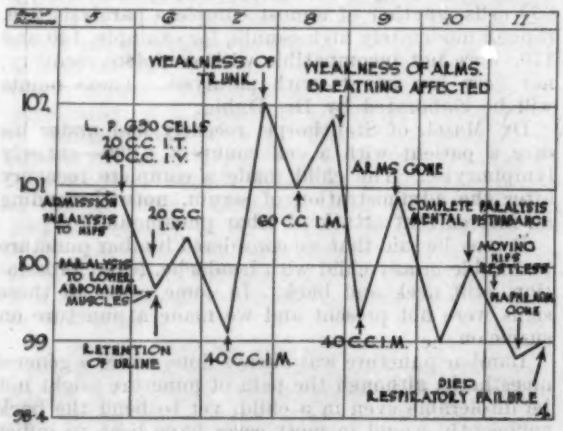


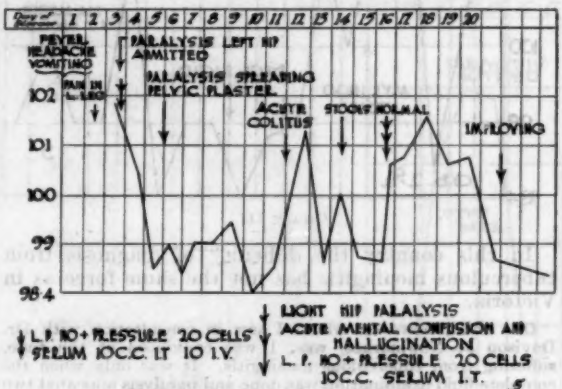
CHART IV.

paralysis of the lower limbs increased and was associated with severe cerebral affection.)

G.J. (Chart VI), aged five and a half years, was admitted to hospital on January 14, 1932 with a not very definite paralysis of the right hip. In spite of serum, 20 cubic centimetres given intrathecally and 30 cubic centimetres given intravenously, his paralysis advanced and by January 16, 1932, was so complete that he could only breathe with his diaphragm and scaleni and had even to have his head turned for comfort. Spinal sign was very marked and the cell count was 270 cells.

There was gradual improvement until May 20, 1932, when he developed bronchopneumonia, in which he was much handicapped by the weakness of his respiratory muscles. He died after three days.

Sometimes this spread was a diffuse and scattered condition with no regular sequence of involvement. (Chart VI.) In a few it took the ascending or Landry form (Chart V), usually fatal, in some without and in the others in spite of serum.



MENTAL DISTURBANCE LASTED 21 DAYS.

CHART V.

In no case did we see what could be called a purely neuritic distribution; distribution was always by spinal segments, as the pathology would lead us to expect, and was seldom completely symmetrical.

Mental disturbance occurred in a few patients, very usually in the fatal cases, but unfortunately in some of the swiftly progressive cases consciousness remained until the end.

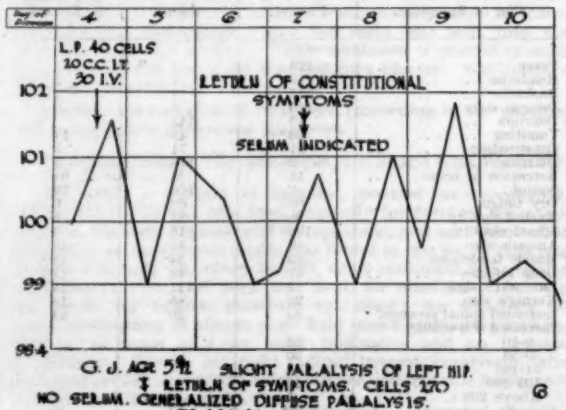


CHART VI.

Fatal Cases.

Altogether there were fifteen fatal cases in the whole series of 128. The infections in these cases fall into two groups: (i) Those of the overwhelming toxæmic type, (ii) those of the ascending Landry type.

Of the former I saw six cases, of the latter seven, and two others, one of each, at the Brisbane Hospital.

Chart IV shows the course of a Landry type of several days' duration in a boy of eighteen years; another lasted only twenty-four hours from the onset of paralysis.

The toxæmic type was more encephalitic in appearance and facial muscular paralysis was present invariably with the cerebral manifestations. It was in three of these that the only examples of squint were found.

An acute toxic case is exemplified in the following:

G.V., a male, aged fourteen years, was seen on December 14, 1931. The onset was two days before with fever, headache, vomiting, abdominal pain, and pain in the neck and back. The day before the patient was first seen abdominal pain increased, much abdominal distension and further vomiting occurred. The bowels were open with a high enema; the abdomen was flaccid. Backache and stiff neck were present.

On the day of admission to hospital the patient's condition was much worse; he was barely conscious. Right internal strabismus was present, with pharyngeal and laryngeal paralysis. He had an "ironed out" face. Respiration was very irregular and purely diaphragmatic. The upper limbs were completely flaccid. In the lower limbs a few weak movements were noted. The neck and spine were rigid. The patient died, without regaining consciousness, from respiratory failure.

The cell count in the Landry type was usually high; in the other quite low, in one which lasted only three days it was only 65, and the cells were mainly lymphocytes; in another which had lasted under forty-eight hours the cell count was only 90.

One patient who developed profound cerebral symptoms with delirium and a mental confusion recovered in part, though there is still extensive paralysis (Chart V).

Convulsions I cannot find noted as a symptom of importance. Abdominal pain and rigidity were present in three cases. One patient was sent into hospital for immediate abdominal operation. Another (quoted above), with much abdominal distension, was thought to be suffering from intestinal obstruction.

Three other fatal cases occurred. In two of these death was due to intercurrent infections (acute summer diarrhœa and pneumonia in paralysed children during the convalescent stage). The third death resulted from acute respiratory paralysis. In this instance the infection was apparently mild and the patient died just after puncture was performed and after administration of a small dose of serum.

I trust that this very condensed report of a very extensive matter may have been of interest. Above all, may it prove of assistance to those who will, I fear, be dealing with this disease again this summer.

Acknowledgements.

In conclusion, I must acknowledge our indebtedness to Dr. Jean Macnamara and the Melbourne Poliomyelitis Committee, to the former for teaching, and to the latter (and former, too) for serum. Nor can I let the work of my devoted resident staff at the Hospital for Sick Children pass unappreciated. Without their cheerful and skilful cooperation we could never have dealt with the epidemic as effectually as I believe we did.

References.

(1) Jean Macnamara: "Treatment of Anterior Poliomyelitis", THE MEDICAL JOURNAL OF AUSTRALIA, December 14, 1929, page 838.
(2) Jean Macnamara: Special article on diagnosis, "Acute Anterior Poliomyelitis", THE MEDICAL JOURNAL OF AUSTRALIA, March 28, 1931.
(3) F. M. Burnet and Jean Macnamara: "The Potency of Human Convalescent Serum in Poliomyelitis", THE MEDICAL JOURNAL OF AUSTRALIA, December 14, 1929, page 851.
(4) G. Draper: "Poliomyelitis".
(5) G. Draper: "Significant Problems in Acute Anterior Poliomyelitis", *The Journal of the American Medical Association*, Volume XCVII, October 17, 1931, page 1139.
(6) R. W. Fairbrother and E. W. Hurst: "The Pathogenesis of, and Propagation of the Virus in, Experimental Poliomyelitis", *Journal of Pathology and Bacteriology*, Volume XXXIII, January, 1930, page 17.

A FEW NOTES ON THE SERUM TREATMENT OF ACUTE ANTERIOR POLIOMYELITIS.[1]

By ALEC E. PATERSON, M.B., Ch.M. (Sydney),
Medical Superintendent, Hospital for Sick Children, Brisbane.

AT the onset of the late epidemic my knowledge of serum therapy in acute anterior poliomyelitis was practically nil, and it was therefore necessary to obtain help from Melbourne. The dosage used by us at the Hospital for Sick Children was thus regulated in more senses than one. Nevertheless we and many fortunate children are under a deep debt of gratitude to Melbourne workers for making available 3,000 cubic centimetres of their serum. The dose advised was 50 cubic centimetres, split up into 20 cubic centimetres to be given intrathecally and 30 cubic centimetres intravenously. As far as was practicable we adhered to this, occasionally giving more, but more commonly less.

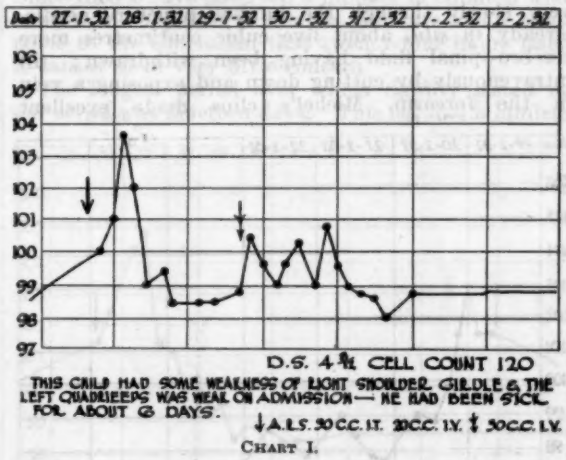


CHART I.

We would have wished for some experimental work, but the threatened dangers to our patients and the small amounts of serum on hand effectively prevented this, and if at times we did err, may I blame the inadequate supply?

To show as clearly and as briefly as possible the end results and the effect of serum treatment as regards the patients under my care I have made a detailed report, a copy of which is given you.

In all 2,870 cubic centimetres of serum were administered to 74 patients, the average dose being

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932.

38·7 cubic centimetres. The results were beyond all expectations. However, I feel sure that had we been able to obtain a larger supply of serum, higher doses would have been given as a rule and our results would have been better.

There is very little to say regarding the actual administration of the serum.

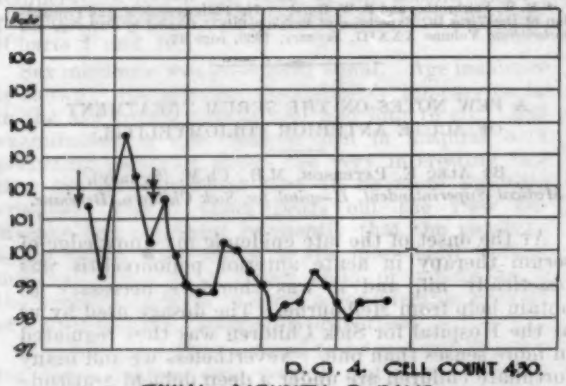


CHART II.

The patient was under an anæsthetic and the dose, estimated according to the time since onset, the child's general condition, and the cell count of cerebro-spinal fluid, was given: (i) At body temperature very slowly through the lumbar puncture needle, already *in situ*, about five cubic centimetres more cerebro-spinal fluid having been withdrawn; (ii) intravenously by cutting down and exposing a vein in the forearm. Michel's clips made excellent

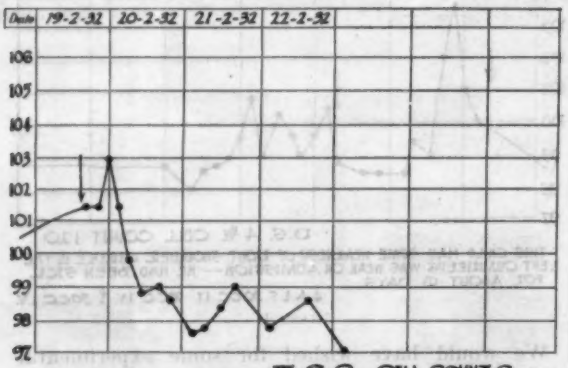


CHART III.

sutures, easily applied and easily removed if another intravenous dose was required. A further dose depended entirely on the child's progress and condition after twenty-four hours. The adoption of the intravenous route was the rule, once only was the intramuscular route used.

The serum produced a profound general reaction in most cases, and in the early days this alarmed us a great deal, but we soon came to the stage of hoping for it.

This reaction began in about four hours and lasted on an average about twelve. The patients were extremely uncomfortable, many told us they had never been so sick in all their lives. Their temperature would rise two to three points, headache was severe

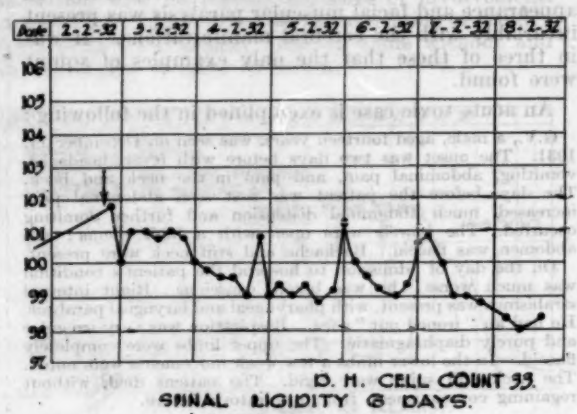


CHART IV.

and their only desire was to be left alone in their misery. Suddenly the picture would change, the child felt a lot better, the headache was gone, food and drink were wanted and from being very irritable, the child showed a pride in attempting to accomplish the various actions asked for by us. Within twenty-four hours there was a considerable drop in temperature, but it was generally two to three days before the temperature actually became normal.

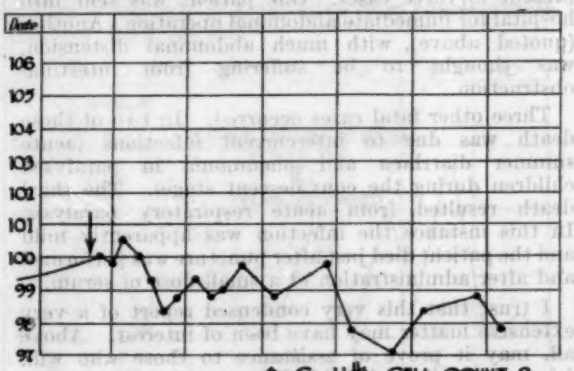


CHART V.

During the next few days the child was carefully watched and tested for any signs of paresis in any muscle groups; if any suspicion of this occurred, all limbs were immediately splinted.

The gradual lessening of the spinal rigidity was carefully noted, this taking on an average about six days. This rigidity was very interesting and the time it took to disappear caused great discussion

during the epidemic, and may cause discussion this evening. Why it occurred I do not know, but I am firmly convinced that neither the lumbar puncture nor the intrathecal administration of serum had anything to do with its persistence.

My proofs for this assertion are as follows. During the epidemic there were admitted to the observation

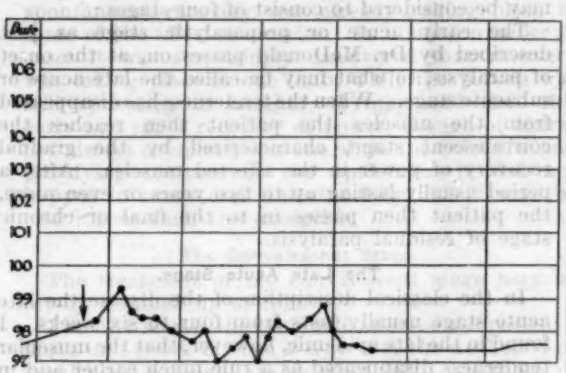


CHART VI.

ward thirty-two suspected patients; as a group there is no mention made of them in the detailed report. They were given a general anæsthetic and had lumbar punctures performed. The cell count in each was low, the average being about two per cubic millimetre. In thirty of these no spinal rigidity was noted at the end of twenty-four hours and it was mainly on this test that we decided their discharge.

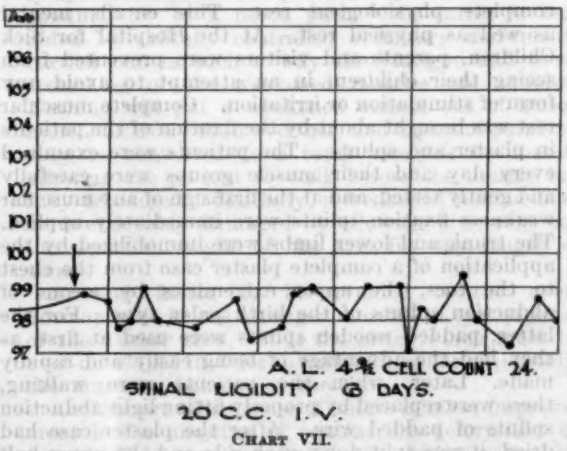


CHART VII.

To my knowledge none of these children developed any weakness or paralysis of their muscles; one of them, however, came back eleven days later with more definite symptoms, and this time received serum and his infection was notified. His symptoms cleared up completely.

Two patients showed a slight increase in rigidity the following day, necessitating further observation. The clinical signs became more marked and a second puncture was done, cell counts of sixteen and twenty-six respectively being revealed. The patients then received serum. The spinal rigidity in each remained for five days.

Then there were other patients in various parts of the hospital during the epidemic upon whom lumbar puncture was done. They were carefully observed

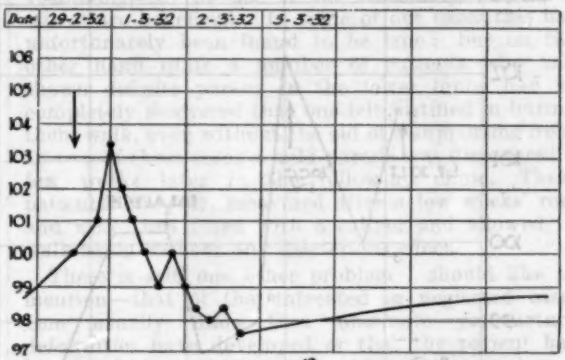


CHART VIII.

and, beyond some pain on extreme flexion, no spinal rigidity could be demonstrated by the next morning. Since then I have observed a number of children who have been subjected to lumbar puncture and I have come to the conclusion that twenty-four hours later it causes them very little concern.

The longest period over which rigidity lasted in our series was nineteen days. This lad had a lumbar

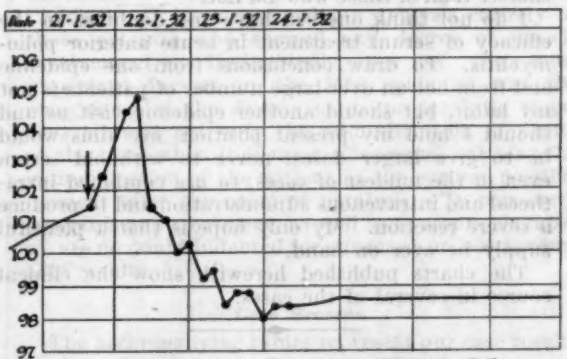


CHART IX.

puncture; his cell count was fifteen; he received no serum. Another in the early days having a low cell count received similar treatment. His rigidity persisted for thirteen days. Both these children became paralysed. One is still an inmate; the other has been discharged completely cured.

From the records of the Mater Misericordiæ Children's Hospital I have learnt that one child

retained its rigidity for seven weeks; serum was given intrathecally and intramuscularly in ten cubic centimetres doses.

Few received serum by the intravenous route only. In this group the rigidity took from five to seven days to disappear.

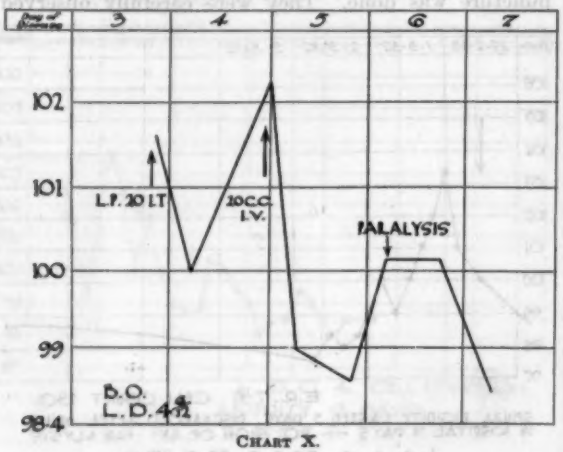


CHART X.

It has been stated that once paralysis has set in, serum is useless. Early in the epidemic we believed this to be true, but it was not long ere we discovered it to be quite wrong. No matter to what extent paralysis is present, as long as the patient is still febrile, I am of the opinion that serum is beneficial. The lives of some of our patients were saved by giving it, in others it appeared to check further spread, and in comparing the paralysed patients the period of detention in hospital of those who had it was shorter than of those who did not.

I do not think one can have any doubt as to the efficacy of serum treatment in acute anterior poliomyelitis. To draw conclusions from one epidemic and from not an over-large number of patients is not my habit, but should another epidemic visit us and should I hold my present position, my aims would be to give larger doses, never to withhold serum even in the mildest of cases, to use combined intrathecal and intravenous administration and to produce a severe reaction. My only hope is that a plentiful supply be ever on hand.

The charts published herewith show the clinical course in several of the cases.

A DISCUSSION ON THE TREATMENT OF ANTERIOR POLIOMYELITIS FROM THE ONSET OF PARALYSIS TILL THE END OF SPONTANEOUS RECOVERY OF POWER IN THE MUSCLES.[1]

By L. W. N. GIBSON, M.B., Ch.M. (Syd.), M.Ch.Orth. (Liv.),
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I HAVE been asked to say a few words on the treatment of infantile paralysis during its late acute and convalescent stages. As the time allotted is so short and as I wish to summarize briefly the results of our series of cases at the Hospital for Sick Children, I must needs confine my remarks to a few of the general principles which govern our methods when treating patients in these stages.

From the point of view of treatment the disease may be considered to consist of four stages:

The early acute or preparalytic stage as just described by Dr. McDonald passes on, at the onset of paralysis, to what may be called the late acute or subacute stage. When the tenderness has disappeared from the muscles the patient then reaches the convalescent stage, characterized by the gradual recovery of power in the affected muscles. After a period usually lasting up to two years or even more, the patient then passes in to the final or chronic stage of residual paralysis.

The Late Acute Stage.

In the classical description of the disease the late acute stage usually lasts from four to six weeks. I found in the late epidemic, however, that the muscular tenderness disappeared as a rule much earlier and in fact in some cases was either entirely absent or too mild to be elicited. When present, on the other hand, the tenderness was often quite marked, and early in the epidemic was a not uncommon cause of error in diagnosis, especially when accompanied by a history of trauma. I remember also a patient whose condition on his admission to hospital closely simulated an acute abdominal crisis, and it was only after careful questioning that a typical pre-paralytic history was obtained.

In discussing the treatment of this phase, one might very truly make use of the time-honoured slogan masterly inactivity. The essential factor is complete physiological rest. This entails mental as well as physical rest. At the Hospital for Sick Children, parents and visitors were prevented from seeing their children, in an attempt to avoid any form of stimulation or irritation. Complete muscular rest was brought about by the fixation of the patients in plaster and splints. The patients were examined every day and their muscle groups were carefully and gently tested, and at the first sign of any muscular weakness fixation splints were immediately applied. The trunk and lower limbs were immobilized by the application of a complete plaster case from the chest to the toes, the upper extremities by means of abduction splints of the birth palsy type. For the latter, padded wooden splints were used at first, as they had the advantage of being easily and rapidly made. Later, when the patients were walking, these were replaced by properly fitting light abduction splints of padded wire. After the plaster case had dried, it was split down each side and the upper half was removed in order to minimize the danger of pressure sores *et cetera*. I should like to add here that before the plaster was applied these children were usually most uncomfortable and miserable, and were continually making attempts to move about in order to get comfortable. Immediately the plaster was applied they settled down and became perfectly quiet and happy, even in spite of excessively hot weather.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932.

When a period, ranging in this epidemic from two to four weeks, had elapsed, the patient passed into the third or convalescent stage, and the time arrived to examine the child carefully in order to estimate the extent of the damage done. In many cases it was found that the paralysis had spread to neighbouring muscle groups, while in others it was found that spontaneous recovery was already taking place in some of the affected muscles. In a small number of cases it was surprisingly discovered that recovery was taking place in one limb, while the paresis was still spreading in another. I may add that this early and rapid recovery of power, though of course not always complete, was one of the most definite and gratifying features of the epidemic. It did not happen in every case unfortunately, but it was the rule rather than the exception.

The Convalescent Stage.

The treatment of the convalescent stage may be briefly summed up as follows.

The first step is the immobilization of the patient's limbs and trunk in such a way that the weakened or paralyzed muscles are placed in a position of relaxation.

The second step is the institution of muscle-training under the supervision of a skilled masseuse.

It is unfortunate that the word massage is connected in so many people's minds with the treatment of infantile paralysis. The essential thing is not massage, but muscle reeducation. Massage, if it be carried out at all, should be confined to gentle stroking and kneading, with the affected muscles maintained in the relaxed position. It may then have a beneficial effect on muscle wasting in the early stages by increasing the blood supply and lymph drainage of the part, but it will not restore conductivity to a nerve path. The latter is best aided by skilfully controlled voluntary movements carried out in such a way that fatigue and over-stretching of the weakened muscles are avoided. Nothing will put a weakened muscle out of action more effectively than over-use, and a partial paralysis may be converted to a total paralysis by merely allowing the patient to walk before the muscle has fully recovered. Similarly, a weakened deltoid may be irretrievably damaged by allowing it to take the unsupported weight of the arm for any length of time.

Another factor to be avoided is the over-development of the stronger muscles at the expense of the weaker ones. It is the latter which must be over-developed in order to maintain, as far as possible, the muscular balance of the limb.

Another prevalent cause of failure to effect improvement is the allowing of contracture deformities to occur. These are usually of the flexion type, the most common being footdrop, flexion at the knee, flexion at the hip, wrist drop, adduction of the shoulder, and last, but not least (that orthopædic bugbear), scoliosis. When contractures of this type are allowed to occur, the weakened muscles are being continuously stretched and very little recovery may be expected till the deformity is overcome.

As regards the later treatment during this stage, one is faced by the problem of when to let the patient walk. This is usually a matter for judgement on the part of the surgeon. Various American surgeons after an exhaustive analysis of numerous cases, treated and untreated, have laid down the general principle that the weight bearing use of muscles with or without irons in the first year is detrimental, and that non weight-bearing exercises (for example, swimming, or the use of the stationary bicycle *et cetera*) are beneficial. In some of our cases this has unfortunately been found to be true; but on the other hand quite a number of patients who had shown definite paresis in the lower limbs had so completely recovered that one felt justified in letting them walk, even without the aid of a supporting iron. In two of these cases a mild paresis was discovered a few weeks later in the follow-up clinic. These patients, however, recovered after a few weeks' rest and were then fitted with a caliper and allowed to walk again without any deleterious effect.

There is still one other problem I should like to mention—that of the untreated or neglected case. One usually finds that obstinate contracture deformities have developed or that the patient has been making valiant attempts to use a weakened limb, with the result that any mild weakness he had has been converted into a severe paresis, or even total paralysis. The position may seem hopeless at first, but it is remarkable how much recovery may be obtained by merely correcting the deformities and splinting the limb in the desired position.

I remember seeing a patient in England who came to hospital for treatment seven years after the onset of the paralysis. He had a marked *genu recurvatum* deformity due to walking with a flail quadriceps. After some months' treatment by splinting at rest and gradual reeducation he had recovered about 50% of power in the paralysed muscle.

Before closing I should like to summarize briefly the results of the cases seen at the Hospital for Sick Children. (I have confined my analysis entirely to those patients who showed any degree of muscle involvement.) Undoubtedly one of the most important factors in the late epidemic was the recognizing of the disease in its preparalytic stage. This allowed not only the administration of serum at the time when it is most efficacious, but also the immobilization of the patient at the earliest possible moment after the onset of paralysis. To this early diagnosis and treatment, as well as to serum therapy, we are no doubt indebted for our more or less favourable results.

Case Records.

The accompanying tables represent our case results in summarized form.

TABLE I.

Summary of Case Results, including only those Patients who showed any degree of Muscle Involvement.

Group.	Number.
Patients completely cured	18
Ambulatory patients (still undergoing treatment)	16
Patients still in hospital	22
Deaths	12
Total	68

TABLE II.

Comparative Analysis of Sixty-eight Cases.

Number of Patients seen before the onset of Paralysis: 14.

Serum.	Completely Recovered.	Still Undergoing Treatment.	Died.	Total.
Given	6	1	3	10
Not given	1	3	0	4

Number of Patients seen after the onset of Paralysis: 54.

Given	6	11	6[1]	23
Not given	5	23	3	31

[1] Two died from intercurrent disease.

TABLE III.

Showing Distribution of Paralysis.[1]

Site.	Number of Cases.	Percentage.
Left lower limb	40	59·7
Right lower limb	32	47·7
Right upper limb	20	30·0
Left upper limb	19	28·4
Trunk	19	28·4
Face	2	3·0

[1] Muscles most frequently affected were quadriceps, deltoid, hamstrings, anterior muscles of foot, and triceps in that order of frequency.

TABLE IV.

Analysis of Deaths.

Cause of Death.	Number of Cases.
Toxæmia in the preparalytic stage	3
Respiratory paralysis within three days of admission	6
Intercurrent disease some months after admission..	2
Unknown	1
Total	12

In a detailed review of the case records three factors stand out as more or less definite from the point of view of prognosis.

The first factor is the cell count of the cerebro-spinal fluid. While for the most part a high cellular content meant a rather severe type of infection, the reverse was not necessarily true. Two of our patients with the most severe infections had a cell count of under 20 per cubic millimetre. (It is definitely inadvisable to wait for a high cell count before giving serum.)

The second factor is muscular tenderness. This was seen to be a much more reliable prognostic sign. Practically all of the patients with severe infections showed marked muscular tenderness, whereas in the milder infections and in those in which recovery occurred most rapidly, this sign was for the most part absent.

The third factor is the age incidence. Although there were actually more younger than older children affected, it is evident that the older patients suffered more ultimate damage. (The average age of all patients in my series was 4·7 years. The average age of those still in hospital is 6·2 years.)

Acknowledgements.

In conclusion, I should like to convey my thanks to the members of the staff of the Hospital for Sick Children for their worthy and untiring assistance during the epidemic.

LATE TREATMENT OF ANTERIOR POLIOMYELITIS.[1]

By A. V. MEKHAN, M.B., F.R.C.S. (Edinburgh), F.R.A.C.S.,
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SUFFERERS from the late stages of anterior poliomyelitis may, for purposes of treatment, be divided into two classes: (i) Patients in whom early treatment has been neglected; (ii) patients who have had adequate early treatment, by splinting and reeducation, for two years.

In all patients belonging to the first class who are seen within two years of the onset of the disease, the error of neglecting early treatment should be amended by putting the paralysed muscles rigidly at rest according to accepted principles. In the case of the trunk or lower limbs this will necessitate recumbency. Light massage, muscle reeducation and prevention of contractures by moving joints daily should accompany the splinting.

If contractures have actually developed they should be corrected by conservative methods, such as gradual stretching and extension. If at the end of six months of treatment none of the paralysed muscles show any signs of recovery, I do not think that any return of power which is worth while is likely to occur, and the patient, for purposes of treatment, automatically becomes transferred to the second class mentioned above.

Of patients of the first class not seen until more than two years after the attack, a few show features which make it still worth while to start as if the attack had just occurred by giving a period of absolute fixation and reeducation. These are cases in which certain groups of muscles show some power in spite of neglect, but are being greatly hampered by lack of support.

The necessity for complete rest in such cases is made more absolute when deformity is developing. A good example of this group would be a patient with active hamstrings and quadriceps, but with a *genu recurvatum* deformity, due to weight-bearing before sufficient recovery had occurred in these muscles. Such muscles often show a striking improvement in power when put at rest, and the rate of recovery is usually rapid when the handicapped muscles, which are struggling hard against great odds, are given a chance by being freed from stretching forces. Here again a good working rule is to persist in rigid fixation while the paresed muscles are showing progressive improvement, but if no sign of improvement appears within six months, further persistence is not likely to be of

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932.

value and, for the purposes of this paper, the patient can be regarded as belonging to the second class.

We now have left for consideration: (i) the remainder of the patients of the first class, namely, those in whom treatment has been inadequate for a period of more than two years, and in whom no special indications for absolute fixation and reeducation are to be found, and (ii) patients of the second class mentioned above, namely, those who, in spite of adequate early treatment for two years, have failed to recover.

Both groups can be considered together, and the only difference between them, in actual practice, will be that almost all members of the inadequately treated group will show fixed deformities, while in the second group, fixed deformities will be a rarety, and the problems confronting the orthopædic surgeon will consequently be much simpler.

We have now to deal then with the treatment of patients suffering from anterior poliomyelitis of at least two years' duration, in whom there is no further prospect of recovery of muscle power. Some of these patients will have fixed deformities, some will have unfixed deformities, many will have combinations of the two, and all will, of course, have paralysis.

What are the objects of treatment? I take it the primary object is to make the crippled patient more contented with his lot in life by giving him a greater measure of independence than this dread disease has left him with. From the point of view of the surgeon, this amounts to taking measures which will lead to increased function.

It may seem didactic to preface any details of treatment by stressing the importance of carefully studying each patient as a whole, and in this I include his mental as well as his physical calibre, before a final decision is made as to the adoption of any surgical procedure.

The fact remains, however, that this can be forgotten: a technically perfect operation may be done on a patient, and the surgeon may achieve his object completely, and yet the patient may be left much worse physically and mentally. This remark applies particularly when the damage is very extensive and of long standing, and the patient has reached early adult life.

Such a patient has become accustomed to his own methods of getting the greatest possible use out of his crippled limbs, and a radical operation such as the arthrodesis of a knee joint, even though it gives independent stability to a limb which was flail-like before, may throw these methods out of gear and leave him regretting constantly the day that he was robbed of the power of bending his knee.

Another useful principle is that the more extensively paralysed a patient is, the less likelihood is there of any substantial improvement following operative interference, and to my mind it is unjustifiable to operate upon any cripple unless the resultant improvement is likely to be really substantial.

I propose now to descend from principles and to deal with the actual detail of treatment in so far as my limited time will allow.

The methods which we have at our disposal are the application of permanent supporting splints and the performance of surgical operations. The ideal to be aimed at is as far as possible to eliminate the use of permanent splints. Unfortunately, there are some patients to whom we can give no useful help. I refer to patients with complete or almost complete paralysis of both arms and legs, with which trunk paralyses are invariably combined. If, however, one arm has good function, a patient with complete paralysis of both lower limbs can be made to walk with the aid of supporting calipers for the lower limbs and a crutch, and, in spite of the cumbersome apparatus necessary, most of the patients welcome the added independence which it gives them. Lock joints at the knee will enable these patients to bend the knees and sit comfortably, and to stiffen the knee in extension for walking purposes.

Before the patient is fitted with supports, gross deformities are corrected; but it must be constantly borne in mind that these patients will always require to wear rather cumbersome supports to enable them to walk. Obviously, then, only deformities which interfere with the fitting and use of these supports, should be subjected to treatment, and such treatment should be as conservative as possible. Extensive operative procedures will be needed only for deformities which prevent the patient from assuming the upright attitude, or which make the fitting of boots and splints impossible. Unfixed deformities in these patients will be controlled in most cases by the apparatus fitted, and only the fixed deformities will require correction. It is cruel and unnecessary, for instance, to perform an extensive stabilizing operation on a "flail" foot in a patient whose knee joint and hip joint are also of the "flail" variety. This patient will require to wear permanently a walking caliper in any case, and this spint will control the foot and render a stabilizing operation unnecessary.

We may now consider treatment in the less extensively paralysed patients—those in whom there is a reasonable chance of enabling the patient to dispense permanently with the use of apparatus. These are best dealt with regionally.

The Upper Limb.

The main consideration to keep in mind in dealing with the upper limb is that the obtaining of the greatest possible degree of mobility is the main object of treatment. If the patient can be made to acquire a small degree of extra voluntary movement, even if that movement is weak, then a lot of effort on the part of the surgeon and patient is justified.

Another obvious principle is that all treatment is governed by the condition of the hand: if the hand is perfectly useless, then the arm is best left alone. If, however, the condition of the hand is good or moderately good, then any procedure which

will enable the patient to place that hand by his own volition into positions which it could not reach before is well worth while.

Fixed deformities in the upper limb are usually not severe or disabling. Pronation deformity is sometimes severe enough to require section of the tendon of the *pronator radii teres*, followed by fixation of the whole arm in the corrected position.

A common condition in this group is paralysis of the deltoid. In such a case if the trapezius is working strongly and the rhomboid muscles fairly well, the operation of arthrodesis of the shoulder joint, with the arm fixed in 90° of abduction (or in adults between 70° and 90°), full internal rotation and 30° of flexion, gives a most striking improvement in function. The active trapezius and rhomboid muscles and any other active muscles inserted into the scapula can now transmit their function to the arm, the operation making the scapula and humerus into one composite hatchet-shaped bone.

In a patient whose elbow flexors are paralysed, but whose wrist and finger flexors are strong, the operation of transferring the origin of these muscles from the inner epicondyle to a higher position, and attaching it to the internal intermuscular septum of the upper arm, gives a useful range of voluntary flexion, and often makes possible the performance of useful actions which could not be done before.

In a case of paralysis of wrist and finger extensors, provided that the active muscles available really have very good power, transplantation of the *pronator radii teres* to the extensors of the wrist, the *flexor carpi radialis* to the extensors of the thumb and index, and the *flexor carpi ulnaris* to the extensors of the three inner fingers, restores with remarkable success the function of which the disease has robbed the arm.

In the hand itself the *opponens pollicis* is frequently paralysed and this greatly impairs function: most of the more delicate functions of the hand depend upon the movement of thumb opposition. Several methods of transplanting available active tendons to the outer side of the base of the proximal phalanx, in order partially to restore this important movement, have been tried with varying success. Steindler transplants the outer half of the split *flexor longus pollicis* and reports good results in seventeen cases.

The Lower Limb.

In the lower limb, the broad principle to bear in mind in planning treatment is that stability is the main desideratum and mobility a secondary one. It is helpful to call to mind that many a patient with a thigh amputation stump of useful length can walk long distances and do heavy work with the aid of a stiff peg leg. A patient with an upper arm amputation at the same relative level, unless very highly gifted in the use of a mechanical arm, gets very little useful function out of the stump.

Automatically, this principle puts out of court as useful measures a great number of the operations of tendon transplantation which have been devised, particularly those about the ankle, unless they are combined with various arthrodesing operations.

Fixed deformities of the lower limb are often severe and disabling.

Flexion deformities at the knee joint can almost invariably be corrected by applying a plaster cast which is cut transversely behind the knee for two-thirds of its circumference. The split is opened and wedges of gradually increasing size are inserted every few days until almost complete extension is obtained. Great care to avoid pressure sores must be taken by thickly padding the front of the knee and the back of the heel and malleoli.

Flexion and adduction deformity at the hip joint can in many cases be corrected by applying extension, but this must be accompanied by fixation of the trunk and opposite thigh in a plaster cast, so as to prevent arching of the lumbar spine and tilting of the pelvis.

Resistant cases will require operative treatment. This consists of an operation after Soutter in which the shortened muscles are stripped subperiosteally from the ilium, and allowed to slide back as the deformity is corrected. The muscles acquire a fresh attachment to the bone in the new position. The method of cutting across the contracted muscles in these cases usually fails, as a very extensive scar forms and contracts, leading to recurrence of deformity. A contracted *tendo Achillis* is best treated by open tendon lengthening. Three notches extending half-way across the tendon are cut on alternate sides, the length of tendon between the notches depending upon the amount of lengthening required. If the foot is now with slight force pushed upward, the notches widen and then the segments of the tendon slide upon each other, leaving a long tendon with continuity of tendon tissues. Healing takes place by tenoblasts and not by undifferentiated fibroblasts, as in a complete transverse tenotomy. The latter operation should not, in my opinion, be performed in anterior poliomyelitis, as non-union sometimes occurs. It is not fair to the patient to take this risk of putting the main calf group out of action when, in many cases of *equinus* deformity, the calf muscles are responsible for the only useful function left in the leg. Early lateral distortions of the foot and early *calcaneus* deformity can often be greatly improved by manipulation under anæsthesia and prolonged fixation, but once these conditions are well established, recurrence is the rule after this procedure and operative correction becomes necessary.

Paralytic Dislocation of the Hip.—Paralytic dislocation of the hip is usually readily reduced and as readily recurs. The so-called shelf operation gives good results and conserves the movement of the hip joint. A substantial shelf of bone is turned down from the side of the ilium above the joint and its weak base is buttressed by a series of small bone grafts obtained from the ilium. As weight is put on the limb the femoral head is prevented from dislocating by this shelf.

Instability of the Knee Joint.—Knee joint instability due to paralysis of the quadriceps in a mild case can, if the hamstrings have full power, be greatly improved by transplantation of the *biceps femoris* and *semitendinosus* to the patella.

In more severe cases where the hamstrings are also inefficient, and where the instability is so great that the patient has to hold the lower part of the front of the thigh with his hand in order to walk, nothing short of arthrodesis of the knee joint will be of any assistance. This operation should not be done unless the patient has a fair degree of power in the muscles controlling the hip joint.

If the operation is done before the patient reaches about twelve years of age, secondary flexion or *valgus* deformity is liable to occur at the lower epiphyseal line of the femur in a certain percentage of cases. In a young adult the advantages and disadvantages of having a permanently stiff knee joint should be placed before the patient and fully understood by him before the operation is advised. Such patients may prefer to wear permanently a caliper with a lock joint, and to obtain the power to bend the knee at will. Interference in the growth in length of the limb after this operation is not a serious factor.

Instability of the Foot.—Instability of the foot may be caused by a flail-like condition in which the foot rolls laterally when weight is put on the limb, or it may be due to fixed *varus* or *valgus* deformity, or to *calcaneus* deformity, with which a marked degree of *pes cavus* is usually associated. The lateral distortions may or may not be accompanied by *equinus* or *calcaneus* deformity.

Flail Foot and Lateral Distortions of the Foot.—The main fact to realize in dealing with flail foot and lateral distortions of the foot is that for practical purposes all lateral movement of the foot occurs distal to the astragalus and none of it at the ankle joint. The main joints responsible for lateral movement and consequently for lateral deformity are the astragalo-scaphoid, the calcaneo-cuboid and the posterior astragalo-calcanean joints.

There are some types of flail foot in which a reasonably good balance is maintained in weight-bearing, and in these cases no treatment is necessary.

If, however, the foot rolls laterally when weight is applied, good stability can be obtained by Whitman's operation. This consists of excising the astragalus and freeing the lateral ligaments of the ankle joint sufficiently to allow the foot to be displaced backward to such an extent that the malleoli ride in the region of the mid-tarsal joints. At the same time the foot is poised in a position of *equinus*.

This operation makes an efficient support of the foot by bringing the weight-bearing line of the limb across the middle of the foot. It also allows the patient to get a purchase from the front of the foot as he walks. Movement is conserved at the ankle joint, and as the foot, in the act of walking, comes up to an angle of 90° with the leg, the scaphoid bone impinges on the front of the tibial articular surface of the ankle joint and locks the foot.

In severe cases of *calcaneo-cavus* Whitman's operation is also very effective, as in this condition backward displacement of the foot is essential, and by Whitman's method, greater backward displacement can be secured than by any other operation. The operation can be beneficially done even in infants, but should not be done in patients older than about sixteen years as it is liable, in these patients, to leave a painful foot.

However, in the less severe cases of *calcaneo-cavus*, the operation used for fixed lateral distortions which is now to be described, is effective, is not as drastic as the Whitman's operation, and does not shorten the limb nearly to the extent that removal of the astragalus does.

In fixed lateral deformities of the foot, the aim of all corrective operations is to excise wedges from the astragalo-scaphoid, calcaneo-cuboid and astragalo-calcanean joints in such a manner that when the freshened bone surfaces come into contact, the deformity is abolished. In addition, a varying degree of backward displacement of the foot is aimed at. The ankle joint is not interfered with.

A plaster cast is applied to hold the foot in the corrected position and is not removed until firm bony fusion has occurred between the astragalus, scaphoid, *os calcis* and cuboid. The soft new bone is further protected by fitting a below-knee splint so as to prevent the risk of gradual giving of the new bone, with partial recurrence of deformity, under the influence of weight-bearing. The support is usually worn for about six months and the patient then walks in an ordinary boot. As this is an arthrodesing operation, involving the removal of a considerable amount of bone, it is necessary to wait until the patient is old enough to develop large osseous centres in the bones to be operated upon.

The size of the centres can be estimated with sufficient accuracy by X ray examination, but it is best to avoid these operations before the age of seven years, and the ideal age is probably twelve years in the average patient. The best methods of doing these operations are those devised by Naughton Dunn and Hoke.

Tendon Transplantations.—Tendon transplantations are sometimes necessary to improve the function of the foot, but are seldom of any value except in association with arthrodesing operations. The reason for this is that the muscle transplanted is unable of itself to stand up to the strain on it, which constant weight-bearing produces.

Transplantation of the *tibialis anticus* to the outer side of the foot in cases of *varus* deformity with marked adduction of the forefoot, if combined with an arthrodesing operation, is the most generally useful. The tendon transplanted must, however, be of full power.

The Trunk.

A severe paralytic scoliosis is always accompanied by marked rotation deformity of the spine. When

well established and accompanied by advanced bony changes this is a rather hopeless condition. Fortunately, the vital structures contained in the chest cavity adapt themselves in a wonderful manner to the altered shape of the chest and even in the most advanced degrees of deformity, pulmonary and cardiac symptoms are rare.

No apparatus furnishes adequate support, but the celluloid jacket and Taylor brace are of definite value, and should be persisted in while the deformity shows signs of progressing.

In the milder cases a period of recumbency on a Bradford frame, accompanied by extension of the head and pelvis, leads to some correction. It should be followed up by fitting an ambulatory brace or jacket, and by exercises designed to improve muscle balance.

Many methods have been devised to improve the deformity by very drastic stretching in special plaster jackets, but the difficulty is that when the process is completed, and the patient is allowed more freedom, recurrence of the deformity almost invariably occurs.

Hibbs, of New York, has for several years been advocating mechanical correction of as much of the deformity as possible, followed by very extensive spinal fusion operations. The method is still on its trial, and unless the final results prove to be exceptionally good, the drastic nature of the methods of preliminary correction and the severity of the subsequent operations will prevent general acceptance of the method by orthopædic surgeons. Fortunately the development of most paralytic spinal deformities can be prevented by prolonged recumbency in a symmetrical plaster bed and care of the trunk muscles during the early treatment. In view of the difficulties which late cases present, special watch should be kept for the slightest early signs, especially during the first two years of treatment.

THE PATHOLOGY OF ACUTE POLIOMYELITIS.[1]

By J. V. DUHIG, M.B. (Sydney),
Director, Brisbane and District Laboratory, Hospital for Sick Children, Brisbane.

IN the course of 1909, there appeared three papers by Flexner and Lewis, of New York, on the experimental transmission of poliomyelitis which, for the first time in English, threw some light on the nature of what until that year had been a most mysterious disease. Since then we have made such progress in the knowledge of the natural history of poliomyelitis that we can mitigate to a large extent its worst results and often completely cure or prevent them. And I think we are within measurable distance of being able to prevent even an attack of the disease in any form or manifestation by means of active prophylactic inoculation.

In their short but historically such significant papers, Flexner and Lewis[1] [2] [3] referred first to the work of Landsteiner and Popper, who succeeded in infecting monkeys by intraperitoneal inoculation of human material and who published their results in German about four months before Flexner and Lewis had made their first inoculation. They then went on to describe for apparently the first time a simple technique for intracerebral inoculation of saline emulsions obtained from the cords of two humans who had died of the disease in the epidemic in Greater New York in the summer of 1909. With the virus "M.A." these authors were successful in propagating the disease through three series of monkeys. They noted incidentally that "not only is the spinal cord active, but the cortex of the brain also" in producing the disease in monkeys. It should be remarked that they had tried in 1907 to transmit the disease to animals, but they had then used only cerebro-spinal fluid with negative results. This fluid does not contain the virus. They concluded that:

> It has long been supposed that epidemic poliomyelitis is an infectious disease. Its mode of spread certainly points to that view. A single successful inoculation with human virus could not establish the view, because the result might be due to a transferred toxic body, but now that successive transfer of the active agent of the disease has been accomplished, any doubt of its infectious origin can hardly be longer maintained.

The second paper, published three weeks later, considerably extended the previous observations. I again quote their conclusions.

> The virus of epidemic poliomyelitis is readily transmissible from man to monkey, and from monkey to monkey, by way of the brain, the peritoneal cavity and the circulation, and that, however transmitted successfully, it becomes established in the spinal cord and medulla, where it sets up characteristic lesions which are followed by equally characteristic effects that exhibit themselves as the usual symptoms of infantile paralysis in human beings.
>
> Moreover, it can now be stated that this experimental form of poliomyelitis in monkeys is a severe and very often fatal disease, and when recovery from the disease takes place there persist residues of paralysis which resemble the paralytic effects also persisting in human subjects of poliomyelitis.

The authors had noted, of course, that the lesions in their monkeys were similar to those in man and that in some of the monkey cords the lesions could be detected by the naked eye.

The third paper of the two distinguished workers, and in a way the most significant, was devoted to a study of the nature of the virus. They reported three cardinal observations, first that they were unable to find associated with the experimental lesions any bacterial or protozoal parasites; second, that the virus was glycerine-resistant to a much greater extent than bacteria, a character common to many other invisible, but none the less active, viruses such as that of vaccinia, and third, that the virus would pass through a Berkefeld filter, evidently a filter which would stop ordinary bacteria.

In these three papers the fundamental nature of the virus of poliomyelitis and the possibility of its experimental transmission was demonstrated for all time.

Since then much work has been done on the disease, but experiment has done little more than confirm the work of the pioneer investigators. This lack of progress is inherent in the qualities of the virus—invisibility and power of infection of a very limited range of species, man, monkeys and with difficulty,

[1] The remarks made by Dr. J. V. Duhig at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932, were based on this paper.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. V. DUHIG.

FIGURE I.
Perivascular infiltration in grey commissure.

FIGURE II.
Perivascular infiltration in anterior horn. Rupture of vessel, probably traumatic.

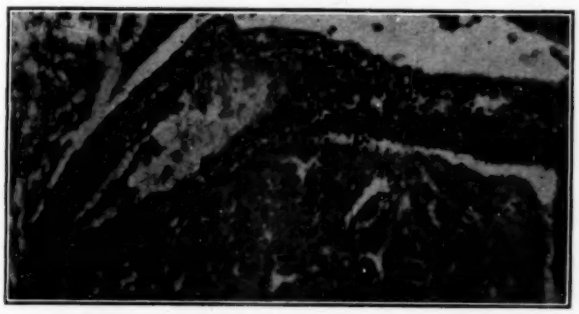

FIGURE III.
Subpial infiltration at anterior nerve root. Infiltration of intrinsic nervous tissues extremely slight.

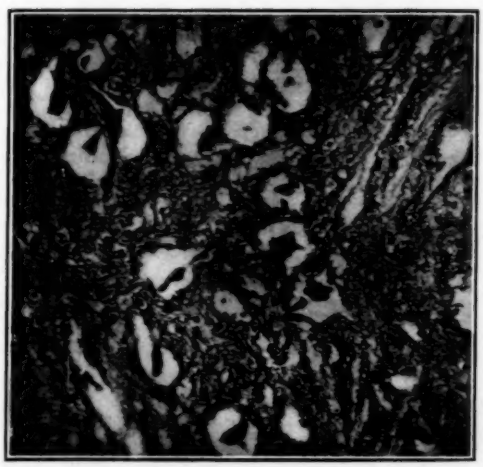

FIGURE IV.
Neurone destruction of severe kind, with very slight exudate or glial reaction.

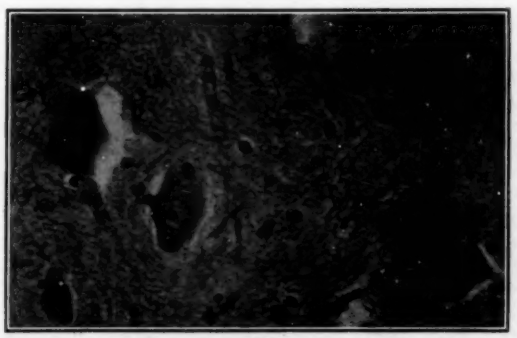

FIGURE V.
Stages in neurone destruction; axones have disappeared. Nuclear changes marked. Cytoplasm becoming granular and hyperchromatic. Satellite neuronophage cells about neurones.

ILLUSTRATIONS TO THE ARTICLE BY DR. J. V. DUHIG.

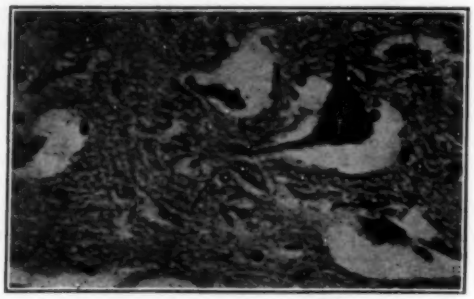

FIGURE VI.
Neurone destruction.

FIGURE VII.
Neurone changes. Neurofibrils coarsely granular. Nucleus eccentric with extrusion of nucleolus. Microglial (spindle cell) reaction.

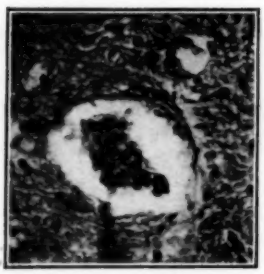

FIGURE VIII.
Neuronophagia — digestion of dead neurone by phagocytes.

FIGURE IX.
"Nodule poliomyelitique"—possibly a replacement gliosis.

FIGURE X.
Early microglial reaction in grey commissure about the central canal.

the horse in which, however, it is doubtful whether the cellular or humoral response is qualitatively similar to those in man and the anthropoids. We do, however, know something of the mode of action of filterable viruses in general and much of the cellular reactions to them, and perhaps more knowledge may be gained by the indirect line of analogy with other viruses than by direct attack. Rivers[4] in a review of the whole subject of the filterable viruses, discussed it from the two points of view, those of pure pathology and cytology. He noted, after reviewing the work of a very large number of observers, that inflammation appears to be, in many virus diseases, a secondary phenomenon, the primary changes being, in all probability, proliferative or degenerative. "In fact", he says, "both types of changes are usually seen, and it is difficult at times to determine definitely whether degeneration precedes proliferation or whether they occur in the reverse order". In whatever order they occur, the changes are later followed by inflammation in the form of exudate, cellular or otherwise. According to the view of many workers, these changes are related in some way to changes in the cells of the organism attacked and the cellular changes are sometimes striking and characteristic, the cells carrying in their cytoplasm or nucleus, or in both, bodies known as "inclusion bodies". According to some views, "inclusion bodies constitute the visible manifestations of a series of activities taking place in living and frequently in growing cells under the stimulating and degrading influences of certain viruses". At the time Rivers wrote, he noted that no significant inclusions had been described as occurring in poliomyelitis and asked whether one is justified in raising the question as to whether inclusions constitute the only characteristic response to this, and, by analogy, to other diseases in which inclusions had not been demonstrated.

Since then, however, Covell[5] and Hurst[6] have done so. Covell in 1930 described acidophilic inclusions in necrosing cells in experimentally induced poliomyelitis, which became fewer in numbers as necrosis progressed. Next year, Hurst confirmed this. He figured nuclear inclusions in the anterior horn cells of monkeys and man. He showed that they occur also in the smaller nerve cells of both anterior and posterior horns. They are few in number, so Hurst found, and on account of the limited time during which they may be found, they are particularly difficult to demonstrate. He notes that, as virus does not reach the cord in appreciable amount, in the experimental disease, until just before the onset of paralysis, the bodies are not demonstrable in the incubation period. Moreover, they are not to be found in completely necrosed cells, but only in damaged ones. In one of the only two cases of the disease which I was able to examine *post mortem*, we attempted to reproduce Hurst's results. When the cord was removed, however, there was no thought of this, and it was preserved in glycerine and remained in that for many weeks before it was subjected to Zenker fixation. The cord selected for study was from the body of a patient who died after a particularly severe attack with a rapidly fatal ending, and consequently very widespread destruction of the neurones of the anterior horns. I was, however, able to demonstrate the presence of nuclear inclusion bodies, clear cut and perfectly rounded, occurring in bunches near the nuclear membrane. They could be demonstrated only up to a certain stage of degeneration of the cytoplasm, rarely when the nucleus was affected, even slightly, and never at the stage of early karyorrhexis. Hurst finally concludes that as far as his evidence goes, and he examined a fair series of controls, the nuclear inclusion bodies he describes appear to be characteristic of the disease.

It seems in the highest degree unlikely that any other organism of a visible kind will ever be successfully incriminated as the ætiological agent of poliomyelitis. As long ago as 1907 and again in 1909, at the time of their successful transmission of the disease, Flexner and Lewis made a thorough search by cultural and other methods for bacteria, but none that could be viewed as the causative agent could be discovered. In spite of that, numerous observers have reported the discovery of bacterial ætiological agents of the disease. Rosenow, of Rochester, is particularly insistent in pushing the claims of a streptococcus. Fairbrother[7] devotes a paper to a review of this misguided literature and to an overhaul of the whole subject of bacterial ætiology. Fairbrother gives a long historical introduction and an imposing bibliography with his paper, which should be read by those interested. He records that following on the demonstration of the filter-passing power of the ætiological agent by Landsteiner and Levaditi (1909) and by Flexner and Lewis the same year, the claims of bacteria were dismissed and that in 1911 Wickman, in a monograph on poliomyelitis, expressed the view that reference to these organisms ought to be excluded from the literature, while Schreiber (1911) was also of the same opinion. After reading Fairbrother's paper, I cannot resist his conclusions that the cocci infrequently isolated by him from the tissues of experimentally infected monkeys have no ætiological relationship with the disease. These organisms are similar in type to those of Rosenow and others and are, probably, the same. In any case, I do not believe they or any other coccus have anything to do with poliomyelitis.

A recent paper, however, by Long, Olitsky and Rhoads[8] revives interest in the "globoid bodies" originally described by Flexner and Noguchi in 1913, and cultivated by them from tissues of the central nervous system from human and experimental sources of poliomyelitis. In the course of an attempt to cultivate the virus of the disease *in vitro*, Long and his coworkers made two very significant observations. The first was that it appears as if virus is capable of multiplication *in vitro*, since, as an example of the sort of result obtained, a tenth transfer "culture", representing a $1{\cdot}3\times10^{-18}$ dilution of the original cultured material, could produce the pathology and signs of poliomyelitis on inoculation into monkeys. It is hardly likely that sufficient of the original material could be carried over to produce the effect observed. The second observation was that this infective tenth transfer was from a culture tube from which it was possible to demonstrate "globoid bodies", while a tenth transfer from a tube containing no such bodies remained non-virulent. This work, so far as I know, has not been repeated or confirmed.

If it turns out to be a fact, it should be of much assistance in the future production of either therapeutic serum or of attenuated prophylactic virus. Were such promise fulfilled, it would be much relief from the present cumbersome and costly method of propagating the virus by animal passage which is the one on which, at present, we are forced to rely.

Biologically the virus shows considerable variation in virulence, as tested in the monkey. The demonstration of this quality, however, was confused by the fact that when filtrates were used, a certain proportion of the virus was left in the filter. That this was so is shown by the fact that the incubation period of the experimentally induced disease was longer with filtrates than with crude cord emulsions. The exact porosity of the filters used was also a matter of some doubt. We now know that with decent technique the virus will pass a Berkefeld V filter, but not a Berkefeld W or Pasteur-Chamberlain 2.

The virus is extremely resistant to physical agencies. In the cold it will remain active for months or years. It is, however, rapidly destroyed by heat, 60° C. for twenty minutes. It resists drying. It is best preserved in 33·3% to 50% glycerine in the ice chest.

The difficulties inherent in the nature of the virus which beset the experimental study of the disease are reflected in the difficulties which have surrounded attempts to explain its pathogenesis. It was originally noted by Flexner that there was general agreement as to the infectious nature of the disease and the fact of the epidemiological nature of the disease in general suggested beyond doubt that the disease was communicable, but since the virus is demonstrable by technique and methods so difficult to apply in field hygiene practice, the pathogenesis, though capable of indirect demonstration, still remains to some extent doubtful. Evidence, however, is accumulating that direct contact between patients or contacts and susceptibles is the main channel of propagation, though the precise form of contact and the precise vehicle still remain in doubt. In addition to this way, the disease can be communicated definitely by foodstuffs, particularly milk. There are possibly other ways. It is of interest to consider the evidence. Some of the best work on the epidemiology of poliomyelitis has been done by Aycock. In 1928 he[9] produced statistical evidence of transmission by contact by a study of the age distribution. He stated that even then, after twenty years of study, there was still uncertainty regarding the mode of spread. The theory of the ingress and egress of the virus through the nasal mucosa had received strong confirmation in the experimental disease and had pointed to spread by unsuspected infections, either through healthy carriers or abortive cases, but the abortive cases had not been demonstrated to anything like the extent that would have been expected from a study of the crude incidence of the disease itself. Again it was possible to demonstrate neutralizing substances in the serum of persons known not to have passed through a frank attack. This suggested an analogy with diphtheria, from which all but a very small proportion of adults are immune by reason of subclinical inoculations of the toxin in early life. But, again, it is a practical impossibility to determine the extent of the immunity in the general population. But an analysis of the incidence on age-groups in a series of epidemics should give strong evidence of immunization in earlier life. This Aycock actually found to be true. He showed from figures taken out over 1912-1925, inclusive, that the age distribution of poliomyelitis in the population he studied showed a preponderance in childhood, the incidence of the disease declining rapidly with increasing years, while the disease in rural populations was distributed more evenly in the older age groups. The age frequency in the rural groups is conditioned by the fewer opportunities of contact in a dispersed than in a concentrated population. Charts taken out for measles, diphtheria and poliomyelitis have in general an age distribution of the same order—a preponderance of cases in childhood and the downward trend with increasing age in all three is more rapid in urban than in rural populations, showing that the concentration of population has an identical effect on the age distribution of the three diseases, so that, as Aycock observes, the mechanism involved is identical for each of these diseases. He concluded, therefore, that the virus of poliomyelitis attains a distribution equal to that of measles and diphtheria, in the course of which the majority of persons become immunized either subclinically or through unrecognized attacks of the disease. And the evidence for this spread by contact is very strong. Stocks[10] has recently reviewed the data for Lancashire over 1920-1929. He says that immunity to poliomyelitis has undoubtedly become widespread in England, and explains it theoretically by reference to the Swedish experience, which shows that villages in which only two or three cases were notified during the 1905 epidemic almost invariably escaped the second, 1911-1913, which picked out those districts and localities missed by the first one. His conclusion, based on an analysis of 172 cases (80% at five years and under) is that, where the disease is endemic, 100 persons acquire an immunity, by mild unrecognized infections, to every one who develops paralytic symptoms and that by the time adult age is reached the majority of people in an endemic area have thus become immune. In epidemic conditions, infection pressure is, of course, unusually high, but it is very rare for an epidemic, however intense, to attack more than 5% of any local population. Further valuable evidence on this point has very recently been published. Paul and Trask[11] attempted an experimental investigation to throw some light on the vexed question of what types of disease can clinically be regarded as "abortive". They did succeed in doing this to a great extent and incidentally strengthened the position we have been trying to establish. From actual cases, contacts and cases of minor illness in association with poliomyelitis during an epidemic, they took washings from the throat and these they injected intracerebrally into monkeys. Out of twelve attempts, two succeeded, and considering the experimental difficulties this was a good result. They put out this added evidence in favour of the belief that certain types of minor illness which accompany poliomyelitis probably represent mild cases of the disease.

It may be legitimate to add to this conclusion that it also strengthens the belief that one of the paths of invasion of the body is by the naso-pharynx and that the abortive types and subclinical carriers are responsible for spread of the disease in part.

Aycock[12] had previously shown that the disease may also be conveyed by milk. He noted that two local epidemics in New York State had been traced to this source and gave a detailed analysis of the Broadstairs (England) epidemic of 1926, comprising 75 cases. Aycock shows fairly conclusively that the majority of the infections were simultaneous and that 14 of the 75 cases could be excluded from contact with an infected person; four patients who left the epidemic area before the epidemic started helped to fix the incubation period accurately, and altogether since milk was the only reasonable common factor, it must definitely be incriminated. One peculiar feature of this outbreak was the unusual age distribution, there being only ten patients under five, and fifty in the two five-year groups, five to fourteen. Aycock's comment is that this is more nearly typical of the age distribution of poliomyelitis in small communities.

In view of the symptoms often associated with a true or abortive attack, such as vomiting and diarrhœa, it is possible that the virus gains entrance through the digestive mucosa. Dr. Sawyer once told me that he knew that poliomyelitis virus had been isolated from the fæces of patients in an epidemic in San Francisco.

We know that it is possible to infect monkeys through the brain, circulation, nose, peritoneum, subcutaneous tissues and peripheral nerves, the sciatic in particular, through which not only the same, but also the opposite side of the cord has been infected. All these paths may be available in the human, but so far our knowledge is scanty and does not extend much beyond that which I have indicated. Flies have been incriminated as vectors, and if the virus is present in the stools, that may be possible, but the evidence is not good.

Sawyer and Herms[41] were unable, experimentally, to confirm the work of others on the effectiveness of the stable fly as a vector of the disease.

Levaditi, Schmutz and Willemin[13] made a careful epidemiological and experimental pathological investigation into the epidemic that occurred in the summer (June-September) of 1930 in the department of the Bas-Rhin (Alsace). They concluded that the hypothesis of propagation of poliomyelitis by interhuman contact agrees best with the facts, but that their findings are not inconsistent with transmission by drinking water (Kling) or by foodstuff (Aycock). After reading their experimental results, one would conclude that they were not dealing with a virus of high virulence; this is unfortunate, in a way, since Levaditi was preeminently equipped to produce experimental results of very considerable value. The findings are rather of a negative type. For instance, while they constantly found the cord virulent, they were unable to demonstrate virus in the cerebral cortex. They established that centrifugal diffusion of the virus along the peripheral nerves does not appear to occur. They demonstrated the virus in the tonsil of one child on the fourth day, but could not constantly demonstrate it in the secretions of the nasopharynx nor in the fæces. But they think the virus must exist in the nose and throat much more frequently than the results appear to show. They did not find the organism outside the human body, in water and milk for example, nor do they think that mosquitoes play an effective part in transmission of the disease.

Marinesco, Manicatide and State-Draganesco,[14] having investigated the epidemic in Roumania in 1927, came to definite conclusions as to the portal of entry and propagation of the virus. They found alterations about the pharynx and small intestine, consisting of an inflammatory process and lymphoid hyperplasia along with the presence of numerous eosinophile cells. This process, they think, would tend to prove that the virus having penetrated the digestive system, after a time is propagated along the lymphatic spaces of the nerves to the spinal cord. This observation should accord with the successful propagation of the disease by intraperitoneal inoculation by Landsteiner and Popper. But in the Roumanian cases no sign of involvement of the micro-sympathetic ganglia in the wall of the gut could be found, as should have been the case if the theory were correct, since the ganglia should be the very first stage. These authors state they cannot support the idea that the intestinal mucosa is the portal of entry. They definitely state their opinion that it is at the level of the upper respiratory tract that the virus enters the organism.

The disease is in all probability conveyed by droplet infection and most often the route of invasion is by way of the nose or throat. From this point there is invasion of the central nervous system. Until the virus penetrates this far, the actual pathogenesis is still surrounded by some doubt, since experimental infection is impossible in the human and difficult in the monkey. But once the virus is in the central nervous system, there is not now much doubt as to the course of events. They have been followed in the admirable experimental studies of Hurst and of Hurst and Fairbrother.

In 1930 Hurst and Fairbrother[15] undertook a study of poliomyelitis produced experimentally in the monkey. At the outset they reported that they examined the common assumption that the spread of infection in the neural axis was supposed to be by the meninges and a primary meningitis to be the first histological lesion. Their earlier experiments did not confirm this, but suggested that the meninges became infiltrated secondarily to the inflammatory process in the cord. They used intracerebral inoculation all through this first series of experiments. Five or six days after inoculation the first sign of muscular weakness is seen in one of the lower limbs, the virus having reached and produced lesions in the lumbar segment of the cord without giving any sign other than a rise of rectal temperature of any changes in the body. It was found on histological examination that all parts of the cord are involved at approximately the same time when the symptoms are compared with the findings, and it was also found that virus could be detected in the cord at the first appearance of symptoms, but not on preceding days. The

authors state that the cerebro-spinal fluid takes no demonstrable part in the transfer of virus to the cord. In a second series of experiments, intranasal inoculation was tried. This series showed that virus does, though not readily, reach the central nervous system through the nose. In one monkey which developed fine weakness and tremor in the left arm, virus was found in the left olfactory bulb and tract, but not in the left frontal lobe and right olfactory tract. It could not be demonstrated in the lumbar cord nor in the cerebro-spinal fluid, though the former showed histological evidence of early involvement. Though the experiments were not conclusive on one point, they do, however, suggest that the path of spread is directly in the nervous substance to the cord, and not by the meninges, since the meningeal involvement was scanty in one animal developing the disease. An analysis of the symptoms induced in the whole set of series shows that in the monkey, as in the human, the nervous symptoms are frequently referable to one or both lower limbs, and may often be confined to these extremities. And histological examination shows that, at the time of the onset of symptoms, lesions are present at all levels of the cord, although paralysis is nearly always complete in the legs, while power still remains in the arm muscles. This is quite evidently not due to involvement by ascending infection, but to special susceptibility of the lumbar cord. With an overwhelming infection all segments may be involved with little difference in the times at which the various muscle groups are paralysed, but with weaker strains of virus, greater resistance of the cells of the cervical cord is apparent. This involvement of the muscles supplied from the lumbar cord after intracerebral inoculation in monkeys before other groups shows how impossible it would be to accept the symptoms in human cases as evidence of entry of the virus by the intestinal tract, as has been suggested. In discussing their work, Hurst and Fairbrother make some very significant observations. Taking up first the question of meningeal spread, they suggest that the meningitis which so very often accompanies the lesions in the cord is only a secondary phenomenon; and suggest as an explanation that as the normal direction of flow of the cerebro-spinal fluid is along the perivascular spaces from the centre of the cord outwards, any inflammatory exudate occupying these spaces must, as a matter of course, be swept into the pial meshes. Meningeal infiltration may mean nothing more than a drainage of cells from the interior of the cord. The findings were definite; meningeal infiltration, at least as far as the brain stem and the cord are concerned, does not occur until the perivascular infiltration beginning in the deeper vessels reaches the surface. And again it is stated that repeated failures to detect the virus in the cerebro-spinal fluid strongly suggest that the meninges do not play an important rôle in the pathogenesis of the disease. Histological considerations also make it unlikely. After intracerebral inoculation into the occipito-parietal region, lesions appear first in the homolateral thalamus, then in the hypothalamic nuclei, the tissues around the third ventricle and the midbrain. The pons is next involved and almost immediately afterwards foci of inflammation appear simultaneously at many levels of the medulla and cord. That the main route of transmission of the virus is the nerve fibre is suggested by this consideration and many others. For instance, when weakness appears first in one leg, it is nearly always on the side opposite to that of the inoculation, showing that some decussating structure is involved. Other relevant matters are discussed in their paper, such as the time factor and upward spread of virus in the cord and brain and the peculiar selective sensitivity of the cells of the lumbar cord. The main conclusions of a searching piece of work are that, from a primary focus of implantation in the cerebral cortex, spread of infection to the brain stem and cord is by way of the axis cylinders, and meningeal spread, while possible, is relatively unimportant, though it probably plays some part in the local dissemination of the infection around the site of inoculation. Meningitis is therefore not the primary lesion of the disease, which begins as a primary degeneration of nerve cells accompanied by inflammation in the interstitial tissues. If, then, the virus travels chiefly by way of the axis cylinders, its appearance in the cerebro-spinal fluid would not be expected, nor, in fact, is it found there except on rare occasions.

A further contribution from Hurst[16] confirmed these conclusions and amplified experiments made with intrasciatic nerve inoculations. Two groups of experiments showed a very definite sequence of events in the infection of different regions of the central nervous system after intrasciatic inoculation, as follows: lumbar cord, leg area of the motor cortex, cervical cord, arm area of the motor cortex. Further, the motor cortex corresponding to the inoculated nerve is infected before that of the opposite side, confirming the suggestion that a decussating mechanism is involved in the transmission of the virus. The experiments further showed that, at an early stage, not only are the lesions invariably more marked in the same side of the cord as the nerve inoculated, but that the next region of the nervous system to be affected is the right motor cortex. This infection of the part of the motor cortex corresponding to the affected leg takes place before the cervical cord shows either virus or lesions. As Hurst says, no theory of the pathogenesis of poliomyelitis in terms of the cerebro-spinal fluid only can provide a reasonable explanation of these observations, so that the conclusion must be that the axones are the main transmitting structures in the disease.

Jungeblut and Spring[17] carried out intracerebral inoculation of virus into two monkeys in which they had previously carried out complete transsection of the cord at the level of the first lumbar vertebra. One of the animals developed on the seventh day typical poliomyelitis, with progressive paralysis of the muscles of the arms and back. Transfer of its cervical cord to another monkey resulted in typical poliomyelitis in that animal, while transfer of the lumbar cord produced no such result. Histological examination revealed typical lesions in the cervical and thoracic cord. The lumbar cord appeared normal. Since there was full cerebro-spinal fluid communication between both fragments of the cord, the conclusion is almost inevitable that virus ordinarily travels along the nerve tracts.

These authors go on to state that in their experience the occurrence of virus in the cerebro-spinal fluid is rare, and that in almost all cases the contralateral limb is the first to show paralysis after intracerebral inoculation.

Hurst[18] next investigated the propagation of the virus by intrathecal inoculation, since it had been well recognized that poliomyelitis had been produced in this way. He succeeded in confirming this view, and the experiments once again showed the axonal spread. He agrees that in man the first step in the introduction of the virus into the nervous system is still doubtful, though he says it is extremely probable that invasion takes place from the nose or throat. However, the virus, once admitted, can be transmitted to other parts of the central nervous system without the intervention of the cerebro-spinal fluid, but that, of course, infection can occur after it is infected. Applied to man, the findings go to show that as virus has never been demonstrated in the cerebro-spinal fluid of humans, the axonic route is probably the path of choice in man as in the monkey.

The actual lesions in man and in the monkey are also remarkably similar. I take the description of the experimental lesions from Hurst,[19] who based his findings on his work on monkeys, already referred to.

Before going on, however, it may be well to discuss the particular meaning we are going to attach to the word "inflammation" in this connexion. Poliomyelitis suggests at once an inflammation of the grey matter of the central nervous system. Are we to use the word inflammation in the broad sense of invasion of tissue by an infective or at least harmful agent, or in the strict sense of a certain set of phenomena, vascular change, exudate, fluid and cellular (of cells, that is) derived from the blood and from the fixed tissues and the cycle of change in the reverse order we think of when we speak of resolution, organization and repair and so on? In inflammation, as we ordinarily think of it, the major phenomenon is the cellular exudate. In poliomyelitis the reaction of the tissues is not at all, primarily, of that kind. To anticipate a little, the reaction is primarily of the kind Rivers has described and to which I referred to at the very beginning. So that if we are to use the word "inflammation" at all in this connexion, it must be in the same sense only of successful entry of an infective or harmful agent. The old classical idea of the changes that form the process of inflammation will not cover this condition, which is a morbid process of quite a different order. A description of the lesions will make this clear, keeping in mind the general facts mentioned by Rivers.

A naked eye examination of the central nervous system reveals, as a rule, considerable pial congestion. This was most marked in one case I examined, the cervical and lumbar enlargements being specially involved, nor was the congestion confined to the cord, but was present all over the brain. In the other case I saw the lumbar and cervical enlargements were congested, but the change was confined to the anterior aspect of the cord. The brain was again congested. The interpretation which should probably be made of this appearance is referred to above, and this aspect need no longer concern us. The lesions may be divided into three categories: 1. Perivascular and extra-adventitial infiltration. 2. Destruction of the nerve cells and their fibres. 3. Diffuse and focal infiltration of the nervous tissues. I have altered the order given in Hurst's paper so as better to fit in with the illustrations I have made.

1. Perivascular infiltration is limited to the grey matter in the early stages, but may be found in the white matter later on, and may spread to the surface of the cord. It is most marked in the large vessels near the central canal (Figure I) and in the smaller vessels of the grey matter. One is shown in Figure II. This vessel is ruptured at one end in the picture, and much hæmorrhage has resulted. Hurst says that this is seen with some frequency, but may result from *post mortem* trauma.

This perivascular infiltration is not constant and is not a necessary accompaniment of the disease and may, if the strain of infecting virus is very virulent, be insignificant in comparison to the destruction of the neurones. The infiltrate may be slight or may be a broad cuff, many cells deep (Figure III).

This figure shows perivascular subpial infiltration at the point where the anterior horn becomes peripheral nerve, that is, it represents a histologically late period, when the drainage from the perivascular spaces in the cord appears on the surface in the pial meshes. The cells are for the most part lymphocytes; with a variable proportion of adventitial and endothelial elements. Sometimes, in virulent infections and at an early stage particularly, the polymorphonuclear cells may be as high as 10%. Cells resembling the mononuclear cells of the blood, eosinophile cells, but never plasma cells, have also been observed. Except at the site of Figure II which probably represents traumatic *post mortem* rupture, I have not seen any extra-adventitial infiltration of the nervous tissues and this, in fact, is never marked.

2. Nerve cell destruction is worst in the lumbar and cervical regions of the cord, where, commonly, from one-half to two-thirds of the anterior horn cells may be destroyed; sometimes a still greater proportion, or even almost the whole may be irreparably damaged. In the two cords I examined I found practically no normal neurones. Death of these may be incredibly rapid, giving in twenty-four hours from the onset of tremor in the monkey a condition similar to that shown in Figure IV. In the field shown there is not a single neurone to be seen even approximating to normal and, as Hurst points out, the phenomenon of neuronophagia is not apparent, which is to be expected in so overwhelming an invasion. Also in this kind of rapid death, many neurones are not greatly altered in outline, but show only nuclear destruction and hyperchromatic cytoplasm.

With a less virulent invasion, there is a series of changes more gradual and more easily followed.

I quote Hurst *verbatim*. The changes are illustrated in Figures V, VI and VII.

> . . . changes of which the first is central chromatolysis, followed later by complete solution of the Nissl substance. Both nucleus and cytoplasm may now swell and become very pale and the former disappears. More frequently, however, the

nucleus shrinks, stains more darkly than usual and occupies an eccentric position in the cell; the abnormally large nucleolus may be extruded. The cytoplasm stains intensely in some parts, feebly in others; it becomes reticulated and then appear vacuoles, some of which reach a large size and contain polymorphonuclear leucocytes, giving the cell a grossly irregular outline. In Bielschowsky preparations some of the neurofibrils stain more darkly than the remainder, while the intracellular network is distorted and opened up by a series of fine vacuoles; there now seems to be a comparatively small number of much thicker and darker fibrils (Figure VII) which may result from the coalescence of a number of normal fibrils or from a heavier impregnation of a few survivors of the original number, with a wider and more irregular meshwork than normal. Granular disintegration then sets in and all fibrillar arrangement is speedily lost.

As degeneration proceeds, neuronophagia begins. In addition to polymorphs (Fig. VIII), which vary greatly in number from case to case, and a few lymphocytes and glial cells, microglial elements rapidly gather in this situation and assume the leading rôle. By their agency the dead neuron is quickly removed and even after forty-eight hours only a focus of these cells, the "*nodule polymyélitique*" of Marinesco, remains to indicate its previous site. (Figure IX.)

These areas are those of the replacement gliosis which are seen after clinical recovery.

3. Tissue infiltration occurs as a diffuse cellular increase in the grey matter of the anterior horns, in the grey commissure, where it is frequently very well marked around the central canal (Figure X) and often over a large part of the posterior horns; focal accumulations also occur.

Some of these focal groups are to be seen at the site of damaged or disappearing neurones, "neuronophagia" just referred to, some are about vessels, and others which are independent. There seems to be no difference between the cells of the diffuse and focal infiltrations. They are in general of three types, polymorphonuclear cells, lymphocytes and microglial cells.

Polymorphonuclear cells are, generally, relatively more numerous in the early stages and in virulent infections. They are frequently to be found as satellites of damaged neurones (Figure IV) and around vessels. Later, of course, they may drain out into the pia and so into the cerebro-spinal fluid.

Lymphocytes are relatively more often associated with late manifestations and with the milder infections and are often to be found in the central canal.

The microglia undergoes enormous proliferation and usually forms the greater part of the diffuse and focal exudate of the grey matter. The cells show a marked degree of polymorphism; in the early stages they are preponderantly rod-shaped, later they are oval or irregular, according to the shape of the nucleus. They correspond to Wickman's "polyblasts". Somewhat similar lesions are to be found in the other parts of the central nervous system affected, but in the pons, medulla, midbrain and cerebral cortex neurone damage is never so severe as in the cord, while in the basal ganglia death of nerve cells was never observed.

Lesions in other organs do not appear to be typical in the experimental disease. Marinesco and his coworkers (*loco citato*), however, are at some pains to describe the lesions of the lymphoid tissues of the nasopharynx and gut and of the mesenteric glands and sometimes of the olfactory mucosa. In their description of the changes in the central nervous system, they appear to attach much significance to the meningeal and infiltrative aspects. They categorically state that the meningitic reaction is a constant lesion. This may have been so in their experience, but is not a universal finding. When they describe the "character of the lesions at the level of the cord", they distinguish two aspects, "the diffuse, confluent, massive infiltration and the nodular process", and go on to stress the vascular process. I believe this to be a mistaken interpretation, as my figures go to show. Meningitis may be absent and the vascular process may only be a centrifugal and not a centripetal one, as we are accustomed to view the ordinary inflammatory reaction. It will be agreed, however, that the "*nodule polymyélitique*" is probably a replacement gliosis after destruction and phagocytosis of the neurone.

Poliomyelitis, then, is an infective disease of the whole nervous system, specially located in the anterior horn cells, for which particular distribution there is at present no ascertainable reason. The disease primarily affects the nerve cells, and their degeneration cannot be attributed to the accompanying interstitial change. Meningitis is not a necessary part of the disease, and is often not marked in the early stages. The virus is propagated along the axis cylinders of the neurones almost exclusively. The only pathological changes which can be detected clinically are those in the cerebro-spinal fluid, and examination of this is a most important diagnostic procedure. Greenfield and Carmichael[20] give a good description of the changes, which vary with the stage of the disease.

In the preparalytic stages, these authors say, an increase of cells is a constant feature, and they quote counts from two other workers ranging from 36 to 1,980. They quote Peabody as stating that none of his suspected cases which showed cell counts below ten per cubic millimetre turned out to be poliomyelitis. At this stage the protein is slightly increased, and glucose and chloride are normal. The cells at this stage are mostly polymorphonuclear cells.

With the onset of paralysis, fairly high counts are the rule, the lymphocytes being in excess. The protein is slightly or moderately increased. Greenfield and Carmichael go on to say:

In spite of the inflammatory nature of the cellular exudate there is no lowering in the percentages of glucose and chlorides. (First edition, 1926.)

There is much confusion of thought in this sentence. Even if the cellular exudate were strictly inflammatory, that would not necessarily result in a reduction of glucose. There is no reduction of glucose sometimes in the early stage of tuberculous meningitis and in poliomyelitis, in which there is no virus in the cerebro-spinal fluid; it would on the contrary be surprising to find a reduction of glucose. During the second and subsequent weeks, in the period of convalescence, the cell count diminishes rapidly, usually less than ten. Along with this there is an increase of protein, which may be very great and may persist for a comparatively long time, occasionally for over three months. This phenomenon is probably associated with the excretion of the

products of the cell digestion, which goes on after destruction of the neurones. A fine fibrin web, similar to that seen in tuberculosis meningitis, is not uncommon in fluids left undisturbed at room temperature. The experience in this laboratory in the matter of cell counts is set out in the table.

Number of Cells.	Number of Cases.
Less than 10	25
10	7
20	10
40	6
60	7
80	5
100..	16
200..	3
430..	1
Total	80

It is most important to examine the cerebro-spinal fluid fresh. The best, and indeed, a quite practical procedure is to examine it at the bedside immediately it is drawn. If it is allowed to stand, autodigestion of the cells takes place, especially of the polymorphonuclear cells, which are, of course, richer in digestive ferments than the cells of the mononuclear series. One of the fluids in our series showed a very low count after standing nine hours, when, to judge by the stage and severity of the disease, the count should have been very high.

In general the globulin content runs parallel with the cell count. The fact of so large a number of cases having so low a cell count—twenty-five had counts under ten per cubic millimetre—requires explanation.

My explanation is that a child may have poliomyelitis and still have a low cell count in the cerebro-spinal fluid. I should put it that a high cell count is diagnostic, but that a low one does not exclude the presence of poliomyelitis in times of epidemic. If a doctor has, after expert clinical examination, made up his mind that a child is suffering from poliomyelitis, he should not be misled into a contrary opinion by a low cell count. I very distinctly do not agree with the opinion expressed in the book I quoted—that a cell count below ten negatives a diagnosis of poliomyelitis. If we return to the pathology of the disease, it will be remembered that a perivascular exudate may not occur at all, and if it does occur, as is usual, of course, the outpouring of cells into the cerebro-spinal fluid is really a relatively late phenomenon. These facts are reflected in the cerebro-spinal fluid findings in some of our cases. These show, too, that one count may not suffice.

B.G.L. had one cell per cubic millimetre, but showed an increase in globulin. He developed definite muscular symptoms.

A.D.S. and G.St. had an increase in globulin with low counts of eight and six cells per cubic millimetre respectively. Both showed definite improvement on specific, convalescent serum, therapy.

E.W. on February 25, 1932, had four cells and no globulin. The next day he had 26 cells and a marked increase in globulin.

R.W. on February 26, 1932, had one cell per cubic millimetre; on February 29 he had 29 cells.

Both these children were cured of their illness with convalescent serum.

In times of epidemic, to wait for a high cerebro-spinal cell count before giving serum in an otherwise clinically well-defined case is analogous to delaying serum administration in diphtheria until a swab is incubated. We know that the virus has gained entrance to the central nervous system before there are weakness and paralysis and long before there are cells in the cerebro-spinal fluid. The real first signs or symptoms have probably nothing to do with the nervous system at all.

Recovery from an attack, either of frank poliomyelitis or of the abortive type, seems to confer a lifelong immunity. The pioneer work in this field was done by Flexner and Lewis,[21] who showed that recovered monkeys were refractory to subsequent attempts at infection. They next showed[22] that the serum of a patient who recovered could give protection against an experimental infection. In this second paper they announce, apparently for the first time in any literature, the use of the serum of recovered children to protect monkeys. This work was so promising that the use of convalescent serum was tried by other workers. Netter, in Paris, seems to have been the first to try the method clinically in a systematic way. He was followed by Anderson and Frost[23] in America. Though much has been done to promote the use of convalescent serum, a matter to which I shall refer in more detail later, the immunology of the disease has not received anything like extensive study. The reason for this is that the virus is invisible and, so far, at least, not cultivable with constant ease and certainty. All animals, excepting man and the monkey, and possibly the horse, are refractory to infection, and lastly the disease is not so widespread that virus in cords can always be obtained *post mortem*. Much of the knowledge we possess is of the statistical kind, but none the less valuable.

Aycock had noticed the immunological similarity between poliomyelitis and diphtheria, in respect of age and population concentration. He and Kramer,[24] to determine whether the relatively low incidence in infants is due to passive transfer of temporary immunity from mother to child, as in diphtheria, tested the sera of mothers and of their newborn babies for the presence of neutralizing substances. The mixed sera and virus suspensions were incubated for two hours, left overnight in the ice box, and inoculated intracerebrally into monkeys. The serum of nine mothers and of their infants completely neutralized the virus. The serum of one infant neutralized the virus, while the animal inoculated with the mixture of the virus and the serum of the mother of this child developed the disease on the seventeenth day after inoculation. The control animal developed the disease on the eighth day. Aycock and Kramer note that an incubation over fourteen days, when the mode is seven or eight, probably represents partial neutralization, that is, some degree of immunity. The serum of two mothers and their babies did not neutralize the virus. No tests were made to determine the period of duration of the immunity thus passively transferred from mother to child, but, as in diphtheria again, Aycock's previous surveys showed a definitely low degree of immunity in the one to five age group,

and this was confirmed by protection experiments with the serum of children in this group. Though the statistical sample in this series of experiments is small, the results are consistent with similar ones on other samples and with findings of another kind, so that their value is correspondingly high and they definitely show that a high proportion of urban mothers have in their serum immunizing substances against poliomyelitis and their newborn children in exactly the same proportion, with complete correspondence between mother and child. This immunity is only transitory in the offspring and is due merely to passive transfer from the immune mother. So that whatever immunity is possessed by any given unit of the population is acquired and not natural.

The very striking and obvious development of active immunity in susceptible subjects after an attack of the disease encouraged the hope of producing artificial active immunity in animals without the actual disease, with the end in view, of course, of producing unlimited amounts of therapeutic serum. In 1918 Pettit described the production of a serum first of the sheep and later of the horse for use against poliomyelitis. The reports of its efficacy were very conflicting, and on the whole unfavourable. Its use in the Roumanian epidemic of 1927 showed it to be definitely inferior to the human convalescent serum. Until recently only Pettit's, of the heterologous immune sera, seems to have received any attention.

As shown by Fairbrother,[25] down to the end of 1929 there was no good, well controlled experimental evidence that previous work of his kind had succeeded. Horses and sheep had been tried, but, as Flexner and Stewart (quoted by Fairbrother), in 1928, stated, no experimental animal other than the monkey had shown itself susceptible to inoculations of the virus, or had developed specific antibodies on repeated injections of virus-containing nervous tissues. Fairbrother reinvestigated the question, using intramuscular injections into the horse. By this means he was definitely able to produce a serum which contained antiviral properties, as shown by the neutralization test. The use of the serum for therapeutic purposes in artificially infected monkeys gave inconclusive results, but in one case the result was promising. In this connexion, it must be remembered that tests of this kind are not strictly comparable in the human and in the monkey, since the mortality in the former is only 20% at the most, as against nearly 100% in the latter. In any case, the experiment served to disprove the idea that only in animals susceptible to a certain virus is it possible to obtain in the serum by immunization substances which will neutralize the virus.

Further experiments on these lines at the Lister Institute seem to have been successful. An abstract of the report of the Institute in *Nature*[26] is as follows:

> Dr. Morgan has been able to immunise two out of four horses to poliomyelitis and obtain a potent anti-serum. The potency is precipitated with the euglobulin fraction of the serum and can be concentrated . . . This is of great advantage in treatment by intraspinal injection.

Following on this, Howitt[27] in 1932 reported successful immunization of two goats and a sheep. This was done for two years by different routes, intravenous to start with, replaced later by intramuscular, subcutaneous and intracutaneous. Howitt claims to have developed antiviral substances capable of protecting monkeys against infection, both when tested by different methods of *in vitro* neutralization and when used therapeutically or as a prophylactic, in a certain number of instances. Some of this goat and sheep serum was also used on human cases of the disease in the Children's Hospital, San Francisco. The results, as reported by Howitt, Shaw, Thelander and Limper,[28] seem to me to be very encouraging, though the authors themselves would go so far only as to say that "too small a group was treated to justify more than an impression that these serums have clinical usefulness". It is interesting to reproduce the table summarizing the results of this latest attempt at large scale production of immune serum. The table is copied *verbatim* from the paper referred to.

Summary of Results, using Goat and Sheep Serum, alone or combined with Human Convalescent Plasma.

Type of Serum Treatment.	Total Number of Cases.	Paralysis.			Death.
		None.	Transient.	Permanent.	
I. Early—					
Goat serum	4	2	2	0	0
Goat and human convalescent	1	0	1	0	0
Sheep serum	8	6	2	0	0
Sheep and human convalescent	3	2	1	0	0
II. After onset of paralysis					
Goat serum	1	0	0	0	1
Goat and human convalescent	6	0	0	4	2
Sheep serum	2	1	0	1	0
Sheep and human convalescent	5	1	2	2	0

In April, 1930, Weyer, Park and Banzhaf announced the production of a potent antipoliomyelitic horse serum, and in 1931[29] published their results of actual production and of concentration. This latter point is of great importance in view of their other findings as to the route of administration. They summarize their findings by saying that the horse, apparently insusceptible to poliomyelitis, can be stimulated to the production of virucidal antibodies, but that success is not constant. Concentration is possible and raises the potency four-fold. This finding is important in view of the fact that horse antibody preparations were found to be more effective when used intraspinally than even greater amounts directed into the blood stream. These workers took the opportunity of comparing the potency of their horse serum with that of human convalescent serum. They titrated two pools of convalescent serum, each containing serum from sixteen individuals, with a potency of over one to 20 of virus, but less than one to 30. Horse serum could be produced at least equal to this, but concentration increased it fourfold in potency. Pooled serum from "normal" adults had only half the potency of convalescent serum. They finally concluded that treatment was more effective when therapeutic sera are given through the spinal route. Rhoads[30] was given samples of horse serum by Park for testing, and found striking irregularity in the *in vivo* results of attempts at neutralization of virus, though *in vitro* the samples did

exhibit an antiviral effect against a constant potent filtrate virus of poliomyelitis. It could not compare, for instance, with convalescent human and monkey serum for constancy of effect. An interesting result of Rhoads's work is the nature of the relationship between the inactivating substance in horse and human sera respectively. The results raise the question of doubt of the identity of these substances. Antipoliomyelitic horse serum would seem not to be the exact equivalent of convalescent and is hardly therefore, if this is so, a perfect substitute for it.

Besides attempts at immunization with crude virus, attempts are being made to produce active immunity in animals by partially or wholly neutralized virus, in a fashion analogous to immunization by toxin-antitoxin mixtures, following the demonstration by von Behring of dissociation of toxin from such mixtures, of diphtheria, for example, and by attenuated virus, in a fashion analogous to immunization by formolized or carbolized toxin, diphtheria anatoxin for example. Active immunization in this way has the advantage that it does no serious damage and in monkeys, for instance, immune body could be produced in the serum without paralysing or endangering the life of the animal. Rhoads[31] attempted to immunize monkeys with mixtures of poliomyelitis virus and convalescent human serum, in the same way as it had been found possible to protect against vaccinia. The injection mixtures—of much overneutralized virus and serum—were first found to be non-infectious when injected into the brain of normal monkeys, and were injected into the test animals intracutaneously and subcutaneously and, in a separate series of animals, instilled intranasally. The results showed that a certain number of the treated animals acquired protection and in the process showed no symptom of the disease. The intranasal experiment was inconclusive, but at any rate no pathogenic effect was observed to occur from it. Soon afterwards, Brodie and Goldbloom,[32] with a slight variation in technique, and with a different end in view, confirmed these findings. They examined the effect of injecting serum known to possess neutralizing power before, with and after the virus. They found definitely that a combination of virus and convalescent serum does produce active immunity, without symptoms of the disease, when the serum is given at the time of inoculation or within three days before or after inoculation of the virus. Serum given after is less effective than if given before or with the injection of serum from the point of view of serum production.

Stewart and Rhoads,[33] reviewing the methods and results of attempts to immunize monkeys, noted two facts, first, that it is impossible to protect monkeys with killed virus and, second, that definite though inconstant resistance can be brought about by the intracutaneous and subcutaneous introduction of the living virus. Using these methods, they found that the degree of protection conferred by intracutaneous is greater than by subcutaneous injection, and that by neither route was any pathological sign induced.

Though killed virus has not been found effective, Rhoads,[34] using the method of Levaditi and Nicolau for the inactivation of vaccinia virus, was able to demonstrate immunity in monkeys by their resistance to experimental infection and by the neutralizing power of their sera after treatment by repeated subcutaneous inoculations of virus inactivated by absorption on aluminium hydroxide. In the process the animals showed no signs of active disease. Rhoads[35] has, finally, been able to confirm quantitatively the empirical value of the method of reinoculation of virus into monkeys by Flexner and Lewis,[22] so as to increase the potency of the serum. Monkeys which had recovered from typical poliomyelitis were repeatedly reinoculated with large amounts of active poliomyelitis virus and their serum, tested before and after treatment, was found to have increased in potency. The original convalescent monkey serum which had a neutralization value below human serum after reinforcement approximated in value to human serum.

It is interesting to note that the intracutaneous route was used right through this series of experiments, since this route was found by Stewart and Rhoads to be superior to subcutaneous inoculation for giving rise to an active immunity in monkeys.

All these experiments in the production of active immunity in susceptible animals give rise to the hope of similar treatment of humans. This would, I suppose, depend on a large supply of virus and of convalescent serum. The method of choice now, at least in respect of bacterial diseases, is to use a highly potent or concentrated attenuated or killed virus. Until recently it was not believed possible to confer immunity to virus diseases except by using living virus. We now know that the viruses of foot and mouth disease, of dog distemper and of yellow fever can be inactivated by formalin without losing their antigenic power, exactly as is the case with diphtheria toxin. To these may now be added the virus of herpes. Bedson[36] has shown that this virus, treated with low concentrations of formalin, is capable of producing a high degree of immunity in guinea-pigs. If it can be possible to maintain large supplies of living virus by cultural methods, active prophylaxis on a large scale will be of more than academic interest, more particularly if safe inoculations with inactive virus can be made possible. So long as the supply of virus is limited, the response of inoculated animals to immunization capricious and the potency of their serum inconstant, the use of such serum for treatment cannot be trusted to give good results, at least as good as those from the use of convalescent serum. Upon this we are forced to rely and human convalescents are the main, almost the sole, source of supply. Netter, in Paris, seems to have been the first to use convalescent serum in a systematic way with good results, but the obvious difficulties of securing serum seem to have discouraged what is undoubtedly a valuable method of treatment. There is no doubt that, given in the preparalytic stage, it will prevent death and severe paralysis and sometimes paralysis is prevented altogether. The published results of Aycock and his co-workers in America and of Jean Macnamara in Melbourne are quite convincing.

Aycock and Luther[37] published their first observations on the use of convalescent serum in 1928. In

their paper they disarm criticism or perhaps excite it, according to the prejudice of the reader, by saying:

> In our experience the severity of the symptoms, the physical observations and the examination of the spinal fluid during the preparalytic stage are not of sufficient diagnostic value to warrant any opinion as to the occurrence or severity of paralysis.

That is to say, we must take their word for the diagnosis, and count as cured those abortive cases which would not have shown paralysis. This is undoubtedly the weak point of all the statistics on poliomyelitis, especially of those on this form of treatment. My own attitude is that persons of the unchallengeable experience and integrity of Aycock and Macnamara would be much more often right than wrong in a diagnosis of poliomyelitis. How to treat statistically the potentially abortive case I do not know. The following table sets out the experience of Aycock and Luther in the treatment of 106 cases published in 1928.

Group.	Treated (106).	Untreated (482).
Mortality	0·94%	14·0%
No paralysis	36·0%	1·2%
Severe paralysis	5·7%	46·6%

Their modest conclusions were that the effect of convalescent serum was favourable as shown by (i) a low mortality rate; (ii) a low average total paralysis; and (iii) a strikingly low paralysis of the severer grades. Aycock, Luther, McKhann, Smith and Kramer[38] reported in 1929 the result of treatment by convalescent serum of a further group of patients. In this outbreak 116 treated showed a fatality rate of 6%, 297 untreated a rate of 18·5%. An examination of the muscles of 99 of the untreated showed paralysis of the two severer grades to the extent of 30·3%, those of the treated, 3·7%. On every basis of comparison of the two groups, the value of the serum is evident. The authors state that the outcome in the two groups is strikingly different.

Marinesco and his co-workers used convalescent serum, Pettit serum and treatment by X rays. They give their results as percentages of complete cure, as follows:

By Netter's method (convalescent serum)	31·5
By Pettit's serum	23·3
By Bordier's method (X rays)	22·2
No treatment	15·3

The outstanding work in connexion with the disease in Australia has been done by Dr. Jean Macnamara, working on behalf of the Victorian Committee for Poliomyelitis. In conjunction with Morgan[39] she has published a complete review of the work of serum therapy from 1925 to 1931. To these authors I am deeply indebted for a reprint of their paper, and especially to Dr. Morgan for the facilities he allowed me for seeing the technical side of serum production on a commercial scale.

Over the period mentioned, 133 patients received serum treatment, one died; they showed a total average paralysis numerically expressed by the authors as 4·9. Of a total of 227 cases seen during the epidemic in Victoria from November, 1930, to July, 1931, 76 were serum treated with no deaths, 151 received no serum, and of these 14 died. The numerical ratio of total average paralysis was 3·5 for the treated to 45·4 for the untreated.

In the paper Dr. Morgan sets out the whole technique for serum manufacture. My method is almost exactly the same, except that we do not, for example, find it necessary to paraffin the tube leading from the donor's vein to the receiving bottle. This is washed in sterile neutral potassium oxalate solution (10%) and attached moist to an Unger transfusion cannula. Amounts up to 600 cubic centimetres of blood have been obtained regularly in this way. The table set out below, showing our yields of serum, is not comparable with the figures given by Macnamara and Morgan for the reason that they had at their disposal a giant centrifuge of a seven-litre capacity, while we have had to depend on unaided sedimentation, so that we cannot approach Morgan's wonderful 50·8% yield of serum. To improve our yields I am trying the plan of clotting the residue after decanting the plasma after sedimentation and decanting the serum after separation of the clot. This is mixed in with the plasma and any red cells and fibrin present in the serum will come down in the clot after addition of the calcium chloride. One trial run of the method increased the actual yield of that batch by nearly 25%, giving a total yield for the batch of very nearly 40%. As it is thought that antiseptic added to the serum causes disturbing symptoms, we do not add antiseptic of any kind. We are content to rely on careful aseptic ampouling after filtration through an EK special Seitz filter, and have had no contamination of any part of the sixteen and a half litres of serum produced.

For donors we use only persons with an unequivocal history of the disease with residual obvious paralysis and who are free from communicable disease of other kinds. To obtain a serum of high average potency we pool plasma from not less than eight and preferably from ten or more donors. The serum yields are set out in the table. They are divided into two periods: (i) When rapid production was absolutely imperative in the middle of the epidemic in Brisbane in the summer of 1931-1932, when, that is, all donors were thankfully used whether their veins were accessible and outstanding or not, and when, therefore, the technical difficulties were against a high blood and plasma yield per donor; and (ii) a second period, when ample reserve stocks were accumulated and selected donors could be used at necessarily longer intervals. These facts explain the difference in the period yields.

Table of Serum Yields.

Period.	Donors.	Whole Blood Average in Cubic Centimetres.	Serum Average in Cubic Centimetres.	Yield of Serum. Percentage.
Whole	138	378	120	31
First	103	333	106	31
Second	35	508	165	31

It will be seen that in the second period the bleedings approximated to those recorded by Morgan.

We find that in healthy adults a blood loss of 600 cubic centimetres causes very little inconvenience. At the approach of air hunger, indicated by deep inspiration, the bleeding is stopped.

We have had 16 bleedings, giving an average of nearly nine donors per sitting, for an average serum yield of a little over one litre, the exact amount of serum produced in the nine months being 16,650 cubic centimetres. The greatest number of times any particular donor has been used is four.

I have been told that clinically our serum has been at least equal in potency to that produced by Macnamara and Morgan, which we were very thankful to be able to have available at the start of the epidemic.[1]

The most important point in serum production is the one emphasized by Macnamara and Morgan, which I quote.

> Adequate aftercare of all potential donors—paralysed adults and children over the age of 10—in the intervals between epidemics and personal practical interest in their struggle for recovery is the crux of any scheme for the provision of serum stocks in an emergency.

That I commend to the careful consideration of all those interested in the production of serum in a hurry and in adequate quantities.

The route of administration is still a matter of dispute. Howitt[40] reviews the matter and produces experimental results of her own. The route usually recommended is the intraspinal, followed by intravenous injection of a total minimum of 50 cubic centimetres. In children both these routes are difficult and dangerous. Howitt gives statistical and experimental evidence in favour of the intramuscular route. Two reports from clinicians using her serum are favourable to the intramuscular method. In the first report (1928), the results were given for a series of 81 patients, 43 of whom received intramuscular treatment. There was neither fatality nor paralysis among the 17 treated within the forty-eight hours of the onset of poliomyelitis, while the amount of persistent paralysis and the number of deaths increased the later the serum was given in the course of the disease. The authors recommend the method and have used it repeatedly during the past five years. They recommend 50 cubic centimetres for children under seven, and 100 cubic centimetres or more for those older.

In the 1928 epidemic in Manitoba the intramuscular treatment was used almost exclusively on the recommendation of the Medical Research Committee of the University of Manitoba. Of 161 patients reported on, 57 had serum intramuscularly in the preparalytic stage; 93% of these completely recovered, and 7% had residual paralysis. Thirty-three received serum too late to be of value; of these 22% recovered, 45% had residual paralysis, and 33% died. Fifty-four did not receive serum; 26% of these completely recovered, 63% showed residual paralysis, and 11% died. There can be no doubt, then, of the efficacy of the intramuscular route if serum is given early, as it should be in any case. Howitt's experimental use of the intramuscular route in treating infected monkeys definitely tends to show the superiority of this route.

[1] Details of convalescent serum treatment of our cases will be published later.

My own feeling is that the intraspinal route is dangerous, and there is no physiological ground for preferring it to intravenous and intramuscular methods. My principle is to bring the patient under the influence of the therapeutic agent quickly by giving it intravenously and keeping up supplies of it to the system by intramuscular injections. Continuous intravenous therapy is needlessly wasteful, since foreign protein is quickly excreted.

Acknowledgements.

I should like, once again, to thank Dr. Morgan, Director of the Commonwealth Serum Laboratories, Melbourne, for allowing me to see the details of the production of serum on a commercial scale. Without his help our production efficiency would not have been so high. I would like, also, to thank Dr. Dale, Medical Officer to the City of Melbourne, for giving me access to his records showing the successful organization of the Victorian Committee for Poliomyelitis in general and the method of organizing a donor service in particular. My admiration for the work of this Committee and its devoted officers, medical and lay, is unbounded. My thanks are due, lastly, to Dr. Keith Ross and Dr. John Power, who bled the donors for me, and to my assistant in the serum department, Mr. Pitcher, for his careful and devoted preparation work.

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EPIDEMIOLOGY OF ANTERIOR POLIOMYELITIS, WITH SPECIAL REFERENCE TO THE QUEENSLAND EPIDEMIC, 1931–32.[1]

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As I understand that only twenty minutes are allotted to the reading of each paper, I can manage only to deal briefly with the outstanding features of the epidemiology of poliomyelitis.

Since the war, on account of the increased incidence of anterior poliomyelitis, cerebro-spinal meningitis and *encephalitis lethargica*, all, epidemic diseases of the central nervous system, considerable attention has been given to their study and interpretation. Anterior poliomyelitis, the disease in which we are concerned tonight, demands our attention, not necessarily on account of its morbidity, or even its mortality rates, but rather on account of its disastrous crippling after-effects.

The Queensland rates obtained in the recent epidemic agree in general with those of epidemics in other countries. As sporadic cases are cropping up from time to time, for the purposes of computation the epidemic is assumed to have commenced on October 19, 1931, in the Paddington district, a second case occurring in Red Hill a month later. The first case in the country occurred at Toowoomba on November 14, 1931. In all there were 299 cases, of which 127 occurred in the metropolitan area and 172 in the country.

The last appreciable epidemic occurred in 1924-25 when 61 cases were reported in Brisbane; in 1929 there were 22 cases, and in 1930 there were four cases throughout the State.

The morbidity rates for 1931-32 were 39·7 per 100,000 in Brisbane and 28·6 per 100,000 for the rest of the State. The case fatality rate was 14·68%, the ratio for metropolitan deaths to country being as slightly over two to one, probably due to the fact that many seriously affected patients were rushed to Brisbane from the country districts. Generally the case fatality rates range from 10% to 25%. The death rate was 4·5 per 100,000 population.

Sex.—Sex appears to have little influence on incidence; if anything, the incidence is slightly greater in males, 54%; in females it is 46% for the State. In Brisbane the ratio is 50 : 50.

Age.—The age incidence agreed with that normally occurring, the most common period being between one to five years in Brisbane and two to seven years outside the metropolitan area. Thus the percentage of cases in children under school age was 61·4 and 44 respectively.

This shift to an older age in country districts, I consider, is significant and should influence our measures for prevention when the next epidemic occurs.

Ætiology.

Doubtless the question has been put to us "What causes an epidemic of poliomyelitis to occur?" Several theories have been propounded.

The first is the communication theory, which deals with the influence of fast modern means of transport in conveyance and spread of the disease.

The second is the theory of epidemic cycles. As in other zymotic diseases, which have their definite cycles of incidence and relative absence, it is possible that poliomyelitis has its periods of epidemic prevalence and that we in Queensland are in the midst of an epidemic wave of the disease. Recently, in the League of Nations' publication on poliomyelitis, it was pointed out that one epidemic year is generally followed by another. Thus it is quite possible that the disease will break out in those portions of the State where it did not occur in the recent epidemic.

In the third place lately, in epidemiological studies, the phrase "epidemic constitution of influenza" has been appearing. It has been coined by those who would group influenza, cerebro-spinal meningitis, poliomyelitis and epidemic encephalitis as arising from a similar origin. They state that: (*a*) The epidemiological features of the cycle of years within which influenza explodes are different from those of influenza free cycles. (*b*) That preceding influenza there is often a rise in general morbidity of the population; an epidemic constitution develops favourable to influenza; there are early enough often mild and atypical clinical forerunners of the disease, and parallel or allied clinical maladies are seen. (*c*) That there are concurrences, similarities, and interrelationships between outbreaks of cerebro-

[1] Read at a meeting of the Queensland Branch of the British Medical Association on November 4, 1932.

spinal fever and poliomyelitis, and outbreaks of influenza, bronchitis and pneumonia.

Summarizing briefly, if the soil is favourable, the seed virulent and active, and the means of transport effective, then conditions are ideal for an epidemic. We must thus guard the system from physical and nervous exhaustion and other causes of debilitation; control the virus to the best means in our power by gargles *et cetera*, and prevent its spread by quarantine, prevention of crowds *et cetera*.

The disease is considered to be caused by a virus—a filter-passing organism. Noguchi claimed to have isolated ultra-microscopic globoid bodies, but later work has tended to disprove this claim. Infection occurs by the naso-pharyngeal route. The virus has been demonstrated to be present in the naso-pharynx of patients and healthy carriers, and is probably expelled in droplets. Amoss and Taylor (1917) found that nasal washings from adults and from some children contained substances which neutralized the virus of poliomyelitis. The presence of these substances may explain why, though many people must be infected, so few actually develop the disease.

It can be seen then why, when people assemble in any form of crowds, the passage of the disease is aided. Similarly, in boarding schools *et cetera*, six feet should be the minimum space between the centres of beds. Furthermore, the nasal mucosa should not be damaged by the use of strong douches, a mild saline solution douche being all that is required.

The Queensland Epidemic.

In an endeavour to shed some light on the question insofar as Queensland is concerned, information was solicited from members of the medical profession in districts where the disease had been epidemic, and also a *questionnaire* was sent to health inspectors who in time forwarded an excellent set of replies, the gist of which is incorporated in this paper under the following headings.

Cosmic Influences.—An epidemic of poliomyelitis generally breaks out in the summer or autumn in dry warm weather. It will be remembered that the Queensland epidemic commenced in November, when at that time the weather was dry and warm, the peak in the metropolitan area being reached in the week January 2 to 9 and three weeks later in the country in the week January 23 to 29. Subsidiary peaks occurred in both instances after a period of three weeks. The rise was rapid, the decline slow, setting in when the rains commenced to fall, though reports from the country as to rainfall were slightly conflicting.

Dust.—Neustaedter and Thro claimed to have found the virus in the dust swept up from a house in which three different cases of the disease had occurred. However, no confirmation has been forthcoming, and in the recent epidemic no uniformity was obtained in the reports as to the presence or absence of dust.

Transmission by Insects.—Some workers were of the opinion that flies or the biting stable fly (*Stomoxys calcitrans*) were transmitters of the disease, the former passively, the latter by means of its bite. The evidence collected in Queensland agrees with the view that these insects cannot be incriminated, as in many areas flies were conspicuous by their absence.

Food.—There is no evidence that foodstuffs act as a means of conveyance with the exception of milk, which has been shown in one or two instances in much the same way as it does as a means of carriage for organisms of scarlet fever or diphtheria, that is, conveying the virus from a carrier engaged in milking to some susceptible consumers. In Queensland no evidence was forthcoming in favour of this means of spread, as the milk supply to the various cases was most diverse, many families in fact keeping their own cow.

Sanitation.—Good sanitation and insanitation occurred more or less in equal proportions in the homes of the Queensland patients, thus agreeing with the consensus of opinion that sanitation has no influence in the spread of the disease, though it must be noted that Kling, Wernstedt, Petterson and others have been able to demonstrate the virus in the washings from the rectum in the acute stage of the illness and in convalescent patients.

Anthropological types.—Though no data have been collected in the recent epidemic it is of interest to note that Draper wrote as follows:

> The type of child which seems to be most susceptible to the disease is the large well grown plump individual, who has certain definite characteristics of face and jaws, is broad browed, and broad and round of face.

The teeth are particularly interesting. It was noticed that in 50% to 60% of all cases at Locust Valley, the central incisor teeth of the upper jaw were separated by a cleft of varying width. The wide spaced dentition has been a striking feature and frequently involves all the single teeth of both jaws, so that each tooth stands entirely free.

Incubation Period.—Various authorities state that the incubation period varies from two to ten days, the average period being three to four days. There is evidence in Queensland that the incubation period can be one day. Wickman considered the period as from one to four days, basing his opinion on the interval between the dates of onset of the disease in two members of the same family. In the recent epidemic there were quite a few instances of more than one case occurring in the same family.

Mode of Spread.—The distribution of the cases in an epidemic is irregular. Wickman (1907), who investigated the big Swedish epidemic, found that the disease spread along the lines of communication, railways, roads *et cetera*. He brought evidence to show that infection was spread by carriers and patients. Persons suffering from a mild form of the disease *sine* paralysis are doubtless present in every epidemic and are a source of danger. Recently Sir George Newman in one of his reports drew attention to the fact that an epidemic of so-called influenza frequently occurred *pari passu* with that of poliomyelitis.

All these points are clearly exemplified in following Queensland experiences.

1. In the early stages there was a tendency for the epidemic to spread through the metropolitan

area irregularly along a line north and south, marked by the south-western and Sandgate railways, from which line the disease branched out over the city. In the State it was manifest how the disease followed the railway lines, ultimately reaching Mareeba in the north, Richmond in the west, and Quilpie in the south-west. The disease was most in evidence south-east of a line running from Maryborough to Goondiwindi, thus leaving a large portion of the State with a susceptible population and with a danger of the rise of an epidemic there this summer. To obviate any delay, the State Department of Health has stationed supplies of anti-serum at strategic points all over the State.

2. It was noticed that the disease broke out for the first time in fresh communities amongst contacts of persons who had arrived from a heavily infected area. Persons visiting families in which the disease occurred subsequent to the visit suffered themselves to a visitation of the disease in the course of a few days after the visiting, thus demonstrating the infectiousness of the incubation period.

A boy, after camping at the seaside, left Brisbane for a six weeks visit to his aunt in the north. Shortly after his departure at the termination of his visit his young cousin contracted poliomyelitis.

It is possible thus for the infection to remain latent, a point of view subscribed to by many authorities.

Another significant case is the following.

After playing in a cricket match, one of the players took ill with poliomyelitis the following day, March 2. A younger brother of another player in the same eleven, but living in another district and who was not present at the match, contracted the disease on April 5. It was stated in the report that this second patient played with the cricket ball used by the first patient which had been taken home by his brother. Probably, however, the virus was received from the elder brother, who would be a healthy carrier.

Two sisters who had been isolated in the bush many miles from a small western town contracted the disease within a few hours of each other. In the town there had been two cases of infantile paralysis, and many children also had fallen sick, displaying symptoms of some slight febrile upset. No one had been near these two girls, but the weekly mailman who delivered Education Department literature, which these two girls studied, after receiving it one Saturday morning. They were the only two in the family to touch the papers, and the only two to be affected by the disease.

It is of interest to note that the mailman's son had been away from school between February 2 and 15 for two periods, with a day of attendance in the middle, one period due to a sore heel, the other to a febrile illness displaying laryngitis of no severity. Both girls took ill on February 14 within a few hours of each other, the day following the mailman's visit. It is hard to find any other means of transmission than by the mailman. The incubation period of one day is worth noting.

From reports received from medical practitioners and as a result of personal observation, it was noted that, in the middle and towards the end of the epidemic, children fell ill showing the following symptoms: headache, sore throat, vomiting, constipation, and fever, the temperature generally reaching 38·3° C. (101° F.), occasionally higher. Recovery was complete in two to three days.

In households where there were several children and where several cases occurred, there was generally a period of three to four days between the appearance of each case. These cases might be diagnosed as gastric influenza, but quite possibly would be mild cases of poliomyelitis and, as such, a danger to others.

Prevention.—Since it has been found that perfectly healthy persons may carry infection and that patients may be infective in the incubation period, as well as for a week or two after commencement of the disease, it is obvious that, to be effective, quarantine measures must be put into operation immediately the epidemic appears to be commencing. Doubtless these measures will be more effective in the less populated country areas than in the denser populated metropolitan area. Thus I am of the opinion that the country school should be closed, especially in view of the fact that the age incidence in the country is higher than in the city. The school acts as the meeting place of children who would otherwise not come in contact with each other.

In the city it is more difficult to decide, as in Brisbane the policy of closing schools only to the younger children was enforced. In Sydney the schools remained open, but the two cities cannot properly be compared.

All that can be said at the moment is that the evidence accrued would point in favour of schools closing, thereby limiting the infective agencies to the various streets in which are situated the scholars' homes, but further research must be undertaken. There is no doubt, however, that if the school is overcrowded and badly ventilated and children are thus brought into close contact with each other, means of conveyance of infection from mild or unrecognized cases or carriers are essentially produced. Sir Shirley Murphy has shown that in London the increased incidence of diphtheria among children from three to ten years of age first became conspicuous in the year 1871—the year in which the *Elementary Education Act* first became operative; that there is a marked decline in incidence during the holidays, and a subsequent rise with the reopening of the schools (due allowance being made for the incubation period and/or some delay in notification).

It is hoped as a result of our State-wide campaign in favour of immunization against diphtheria the cycle of this disease will be definitely upset.

I would conclude expressing the hope that in time (it cannot come too soon) means will be found for immunizing our young population against anterior poliomyelitis. Meanwhile we must rely on early and efficient diagnosis and notification of cases; early quarantine, disinfection of clothes and premises in which a case occurred; the use of mild nasal and throat gargles; and the early and efficient use of anti-serum, the supply of which should be maintained.

Reports of Cases.

ANXIETY FOLLOWING THE USE OF "AVERTIN".

By E. P. DARK, M.C., M.B., Ch.M. (Sydney),
Katoomba, New South Wales.

IT would appear that any facts bearing on the use of "Avertin" as a basal anæsthetic are of interest. It was

brought to my notice early in 1930 by Dr. C. C. Coghlan, and his routine of administration was carefully followed in the short series of thirty-one cases.

The surgical procedures for which "Avertin" was used ranged from the extraction of a mouthful of difficult teeth up to a hysterectomy, and the time taken in the various operations from fifteen minutes to two and a half hours.

In the first half of the series I used 0·12 cubic centimetre of "Avertin" fluid to each kilogram of the patient's weight, but after that reduced the dose to 0·1 cubic centimetre. Of course, it was necessary to use a little more ether, but that seemed a negligible disadvantage to weight against a greater margin of safety. In every case the patient went to the operating theatre profoundly asleep and remained unconscious of any discomfort for from two to five hours after being brought back to bed. Post-anæsthetic vomiting was absent in all but three cases in the series, and then was not distressing.

From the point of view of patient, surgeon, nursing staff and anæsthetist, then, "Avertin" would appear to be ideal; but in the three cases detailed below grave anxiety was caused.

1. A young woman with active pulmonary tuberculosis urgently needed curettage of the uterus; an inhalation anæsthetic was undesirable, and the surgeon asked me to try "Avertin". Everything went according to plan until the dilatation of the cervix was begun, when the breathing became shallow and the patient deeply cyanosed; next, respiration ceased altogether, and finally the pulse became imperceptible. As soon as cyanosis appeared "Camphydryl", one cubic centimetre, was injected and artificial respiration begun. In a few minutes the heart's action improved, and in a quarter of an hour the patient was breathing fairly well and the operation was safely completed. The end result was perfectly satisfactory, but everybody, except the patient, had a fright.

2. A middle-aged woman had had a gangrenous appendix removed; at the operation it was noted that she had some tuberculous peritonitis also. She later developed a pelvic abscess. The abscess formation was very insidious and the patient was running a moderate temperature for a couple of weeks before a certain diagnosis could be made, and at the time of the second operation she looked as though she had absorbed a good deal of toxin. Only 0·1 cubic centimetre of "Avertin" fluid was used to each kilogram of body weight. During the operation the patient's condition was reasonably good, but on being returned to her bed she became profoundly cyanosed, ceased breathing, and then for a time became almost pulseless. "Camphydryl", pituitrin and strychnine were used, but artificial respiration had to be kept up for three hours before her condition was satisfactory.

3. A young woman was operated on for inguinal hernia. The dose used was 0·1 cubic centimetre of "Avertin" fluid per kilogram of body weight. Everything went delightfully until half an hour after the patient was back in bed, when the same train of symptoms suddenly appeared—shallow breathing, cyanosis, cessation of breathing, pulselessness. The usual routine brought about the usual result, complete recovery.

The nursing staff of the hospital had suffered a third severe fright, so it appeared to be a reasonable thing to give up the use of "Avertin" for a time at least. If the hospital had possessed an apparatus for administering carbon dioxide, I should probably have persevered; but in its absence I did not feel justified in doing so until a large enough mass of statistics should be collected, to prove beyond all reasonable doubt that "Avertin" is at least as safe as ether, even in the absence of carbon dioxide.

DEATH FROM POISONING BY ACONITINE.

By ARTHUR PALMER, M.B., F.R.C.S. (Edinburgh),
Government Medical Officer, Department of Health, New South Wales.

ONE has occasionally met with a case of poisoning by aconite, for example by the liniment of aconite, belladonna and chloroform, but as I do not know of a case of poisoning by the alkaloid aconitine having been recorded in Australia, perhaps the following may be of interest.

The classical case is that of Dr. Lamson, who murdered his brother-in-law by administering to him aconitine in a capsule.

The man, the subject of this note, was a chemist, aged thirty-four years, who had been in America and is thought to have brought the drug from there.

At 8 p.m. he took a dose out of a bottle containing a proprietary influenza mixture and soon afterwards became very ill and asked to be taken to hospital. He was then vomiting, the vomit being blood-stained. On admission to Sydney Hospital he stated that he could not see and that he was "going in the legs".

The medical superintendent kindly supplied me with the following clinical details: His pulse was regular and its rate was 80 per minute; the pupils were dilated, equal and regular. The patient became cyanosed and unconscious and died at 10 p.m. without having convulsions.

The remaining 0·8 of an ounce of influenza mixture showed a small amount of a white deposit. When the mixture was shaken up and tasted, it within a minute produced numbness and tingling of the lips which soon spread to the tongue and pharynx, with a rather profuse secretion of mucus. It was at once assumed that the poisoning was aconitine and as one three-hundredth of a grain is said to have caused toxic symptoms and one-fiftieth of a grain death, the mouth and throat were freely washed with a solution of permanganate of potash. No other symptoms developed, but the unpleasant sensation did not disappear until lunch was taken nearly three hours later.

It is stated in "Taylor" that one two-thousandth part of a grain may be recognized by the taste test and will kill a mouse within a few minutes.

At an autopsy about twelve hours after death, the blood was found to be dark, with very few clots; the mucous membrane of the stomach and upper gut was reddened and covered with much mucus.

The Government Analyst, Mr. Walton, found that the 0.8 of an ounce of the influenza mixture mentioned contained phenazone, acetate of ammonium, together with 1·5 grains of aconitine—the last only partly in solution. He also isolated aconitine from the organs.

EXOPHTHALMIC GOITRE IN A GIRL AGED TWO YEARS.

By EDGAR H. M. STEPHEN, M.B., Ch.M. (Sydney),
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THE patient I am about to describe presents a very complete picture of exophthalmic goitre fully developed at the very unusual age of two years.

The symptoms of unusual excitability, heart hurry, tremulousness and ever-moist skin afford almost as striking evidence of the condition as the prominence of the enlarged thyreoid gland and the definite exophthalmos.

History.

There was no history of hyperthyreoidism in either branch of the family. At the time of delivery of the patient, injections of adrenaline were needed to preserve life. She was considered to have some congenital abnormality of the heart and for the first four weeks it was uncertain whether she would survive or not. At the age of five months, she was said to have suffered from quinsy. Retropharyngeal abscess is a more probable

occurrence at this age. She was fat and well from the age of six to twenty-four months. Her tonsils and adenoids were removed at the age of three years, as she was a "noisy" breather, especially in her sleep.

The patient was three and a half years old when I saw her first, but a photograph taken at the age of two years shows pronounced goitre and exophthalmos. At this age hyperexcitability and increased frequency of motions were the most troublesome symptoms to her guardians. When I first saw her she was 107·5 centimetres (forty-three inches) in height (normal, 95 centimetres or thirty-eight inches) and weighed 17 kilograms (two stone nine and three-quarter pounds), the normal weight being 15·3 kilograms (two stone six pounds). The patient is a fair complexioned child with a high colour. Her breathing is noisy to a degree approximating stridor, compression of the larynx by the massive thyreoid gland apparently being responsible.

Hyperexcitability and irritability were striking. The patient was pathetically apprehensive and difficult to win over. There was a history of restlessness at night, her toes and fingers worked and she frequently cried out in her sleep. She was tremulous in the extreme. In intelligence she was almost precocious and a very companionable person to her relatives.

There was marked tachycardia, the pulse rate being 156 per minute. There was a systolic murmur in the mitral area, fairly localized.

She is always hungry and wanting to eat. The bowels acted frequently, up to twelve times in the twenty-four hours. The motions were pale yellow in colour.

Her complexion was florid. Hyperidrosis had been noted throughout life. Her hands felt moist and hot.

The circumference of the neck measured ten and a half inches at the level of the thyreoid cartilage. The gland was uniformly enlarged.

Progress.

The patient has become friendly with increasing acquaintance and has a more composed manner. She has quieter rest at night, though she still dreams. The motions are less frequent, but she has gained only half a pound in weight in four months. She has gained 3·75 centimetres (three and a half inches) in height in twelve months.

The basal metabolic rate has not been estimated for the obvious reasons that the patient's age and temperament render such an examination impossible.

Reviews.

PROGRESS IN ANÆSTHESIA AND ANALGESIA.

THE "Recent Advances" series of text books has been enriched by the publication of Dr. Langton Hewer's "Recent Advances in Anæsthesia and Analgesia".[1] In recent years anæsthesia has become more and more specialized, while new methods and drugs have been introduced with bewildering rapidity. Some of these have proved to be of great value, while others have been found of limited use, or even to be dangerous. Dr. Hewer reviews them all with discrimination and fairness, and states his firm belief: "that no new method of anæsthesia or analgesia should ever be tried on the human body unless there is adequate reason to think that it is at least as safe as the method which it is proposed to replace". The book is well illustrated, has a good index, and each chapter has a list of references to original papers. It does not aim at being a text book in the art of anæsthesia, and so most of the elementary teaching and description of well known methods have been omitted. It provides a concise collection of the more important advances of recent years; the subject matter is set out plainly and the advantages and disadvantages of new methods are tabulated clearly.

The patient's safety is considered as of primary importance, and therefore emphasis is laid on the employment of the oxygen replacer anæsthetics combined with the smallest possible amount of lipoid soluble anæsthetic. It is shown that nitrous oxide and oxygen alone may sometimes be inadequate for operations demanding full muscular relaxation, unless secondary saturation is employed. Hewer considers that this procedure may throw undue strain on the right heart, and he advises the addition of minimal amounts of ether instead. The treatment of shock is discussed thoroughly and clearly, although no mention is made of blood transfusion.

In an excellent chapter on primary cardiac syncope the author gives a detailed plan of treatment; the success of intracardiac injections is attributed to cardiac stimulation from the puncture rather than from the injected drug. A *résumé* of recent work on *status lymphaticus* terminates with the remark that very little is actually known about the condition, and collapse which seems to be due to this cause must be treated on the lines suggested for primary cardiac failure. The indications for the use of carbon dioxide and the methods of administration are well described. Premedication and the basal narcotics are thoughtfully discussed; their advantages and contra-indications are clearly presented. It is stated that deaths have occurred through giving further narcotics before consciousness has fully returned. A warning is given that the injection of adrenaline in a patient under "Avertin" may cause ventricular fibrillation, as sometimes happens under chloroform anæsthesia. Ether convulsions, which appear to be uncommon in Australia, are said to be more frequent in deep anæsthesia, in patients suffering from septic conditions, during hot weather, and when heated ether vapour is employed. The author discusses the many advantages of endotracheal anæsthesia and shows a preference for the use of gas and oxygen. Local anæsthesia is well reviewed and its limitations are defined. Splanchnic block is stated to cause a considerable fall of blood pressure and to be inferior to spinal block, both in safety and effectiveness. A comprehensive summary is given of the present position of spinal analgesia; and the use of "Percaine" by the Howard Jones technique is advised as the most suitable method. Dr. Hewer considers that it should not be used for minor operations or in those for conditions above the diaphragm. Much sound advice is contained in the chapters devoted to anæsthesia for operations on the thyreoid, thorax and abdomen. For toxic goitres the author suggests a combination of "Avertin" and local anæsthesia, after which a face mask is placed in position so that gas and oxygen may be added if required. It is considered that anæsthesia in labour is best achieved by nitrous oxide and oxygen, or, where that is not available, by the combination of "Nembutal" and chloral hydrate. It is admitted that the last mentioned method may cause some delay in both the first and second stages.

The chapter on pulmonary complications is very thorough and gives many valuable suggestions; it is pointed out that their incidence after spinal analgesia is about the same as with general or combined anæsthesia. Operations should be postponed when the patient has a cold or has recently suffered from an infection. The surgeon is warned of the dangers of prolonged exposure of the viscera to cold, of the undue use of the bridge in gall-bladder operations, and of the use of frame retractors in the upper part of the abdomen. The anæsthetist is cautioned against excessive use of narcotics and the use of irritating strengths of ether vapour; he is advised to employ the gas anæsthetics in combination with field block, which may be supplemented by a minimal amount of ether when necessary. The routine administration of carbon dioxide in the post-operative period is considered to be essential.

Generally speaking, this is an excellent little book which may be read with advantage, not only by the anæsthetist, but by the surgeon, the obstetrician and the general practitioner.

[1] "Recent Advances in Anæsthesia and Analgesia," by C. L. Hewer, M.B., B.S.; 1932. London: J. and A. Churchill. Demy 8vo., pp. 195, with 64 illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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TREATMENT.

WRITING on the epilepsies, the late Hughlings Jackson pointed out that there were two ways of investigating diseases and two kinds of classification corresponding to them, the empirical and the scientific. He illustrated the empirical by likening it to the way in which a gardener classifies plants, and the scientific by the way in which a botanist classifies them. The empirical method to Hughlings Jackson was only an arrangement. The scientific method, however, the classification properly so called, was for the better organization of existing knowledge and for the discovery of the relations of new facts; it had a great, though not a direct, utilitarian value. He stated that the gardener and the botanist were different people, but that in medicine the same person had to make classifications for the organization and advancement of knowledge and to make arrangement for direct utilitarian purposes of daily life. In the course of his training the student of medicine, if his training is adequate, learns that he has to be both botanist and gardener. All diagnosis of disease is a classification, and the student at first probably looks at disease from the abstract viewpoint, somewhat similar to that of Jackson's botanist; later on he gradually assumes the rôle of gardener. If his powers of observation are keen, he may see that even in his teachers either the botanist or the gardener predominates, and he will realize the difficulty of striking a proper balance. Coming to the bedside of his patient, the student will endeavour to make a diagnosis. He will try to classify the disease from which the patient is suffering. He will consider the *milieu* of his patient; that is to say, he will try to assess the influence which climate, occupation and other external factors have had in bringing the patient to his present state. He will take the history of the patient in detail and will try to determine the relative importance of each sign and symptom. Then he will make a diagnosis. It will not be sufficient to label the condition and, as it were, to pigeonhole it. He should be able to visualize the pathological changes which are taking place in the organ or organs affected. His insight into the pathology of the illness will, of course, be of the greatest possible assistance to him later on in estimating the effect of treatment and in prognosis. But the making of a correct diagnosis and a knowledge of the pathology of the disease are not sufficient to justify the inception of treatment. Carrying on the metaphor of the garden, we may say that if the disease is the plant, attention must be paid to the soil. In other words, the patient as a human being needs to be studied. Even with specific remedies like antitoxin in diphtheria no rule of thumb methods can be used. The patient is a person who may react in the most unexpected way to any therapeutic measure. It was here that the "ancient disciple of Æsculapius, the old-time practitioner" had the secret of his success —he knew human nature. A student of the ways of medical men has recently said, and with considerable justification, that not only should all specialists graduate to their special work through general practice, but that all medical graduates should be compelled to spend six months entirely divorced from medicine and doing some form of social work among different types of people. If this were com-

pulsory, medical practitioners would recognize the mental factor in illness and would use the patient's mental outlook as a therapeutic guide; they would treat the patient as a patient and not as a case, and they would meet with more success.

About twelve months ago a series of special articles on diagnosis and on laboratory aids to diagnosis was published in this journal; and in this issue there appears the first of a special series on treatment. Many expressions of appreciation of the articles on diagnosis were received, and it is hoped that those on treatment will be useful to readers. They will be written at the request of the Editor specially for the general practitioner, and will reflect the personal experience of the authors. They are offered to the general practitioner that they may help him on the technical side of his treatment. We have endeavoured to remind practitioners that the technical side of therapy is only part of what is needed; and these articles must be used merely as a starting point, as something which may be adapted to the full requirements of each patient. With these words of introduction we would thank those who have already promised and those who will later on promise to contribute to the series.

Current Comment.

CATARACT.

As every medical practitioner knows, in the usual operation (the extracapsular operation) for cataract, the lens capsule is split to allow the escape of the lens and is left in the eye. There are certain disadvantages; for example, the capsule itself may be opaque; scraps of lens substance remain adherent to the interior of the capsule and are apt to cause opacities; needling is necessary after the operation in many cases. Intracapsular extraction of cataract (removal of the lens with its capsule), when satisfactorily performed, is followed by a far better functional result; but it is a more difficult operation, and is apt to be attended by loss of vitreous humour. The evolution of a generally satisfactory method of removal of lens and capsule intact is the ideal to be aimed at by ophthalmic surgeons. Ever since it became recognized that cataract was opacity of the lens, men have endeavoured to devise some means to this end; even Daviel, in his classical paper on the extracapsular removal of cataract, written in 1745, recommends removal of the capsule when it is thickened and wrinkled. Several surgeons in the eighteenth century recommended the removal of lens and capsule intact. In 1845 Christaen expressed the lens with its capsule by pressure on the upper lid. In 1866 Pagenstecher described a method of lifting out the lens and capsule with a spoon. In 1868 McNamara, an officer of the Indian Medical Service, described a method of intracapsular extraction of cataract; on this modern methods are largely based. During the later years of the past century Wright, in the United States of America, and later, Mulroney, in India, also removed both lens and capsule. Recent advances have been associated mainly with the name of Henry Smith, who evolved a method of intracapsular extraction in the course of an enormous ophthalmological practice among natives in India. His records are remarkable and his results brilliant; but these may be due largely to his superlative skill. It is difficult to estimate the merits of Smith's operation from a consideration of his description; for he is so plainly making a special plea. The fact is that many ophthalmic surgeons, despite their having learnt the method from Smith himself, have not obtained satisfactory results in European practice. Several reasons for this have been suggested: An obvious possibility is that the operators have not been able to obtain the great experience necessary for the acquisition of the requisite skill; another suggestion is that the suspensory ligament holding the lens to the ciliary body is tougher in Europeans than in Indians, hence more difficult to break from its attachments. There is no need to describe Smith's operation here; suffice it to say that all manipulations, after the limbal section and the iridectomy, are made by pressure from without; a great deal of skill is necessary on the part of the operator and his assistant. More recently Barraquer has devised an operation in which the lens and capsule are drawn out by means of an instrument that is fixed to the front of the capsule by the action of an ingenious vacuum pump. Barraquer's operation aroused a great deal of interest, but has met with little favour. The Stanculeanu-Török-Elschnig operation, the latest method of intracapsular extraction, is discussed in a recent paper by G. Joseph Gnanadickam, an ophthalmic surgeon attached to the Swedish Mission Hospital at Tirupatur, India.[1] Stanculeanu and Knapp seized the anterior surface of the capsule with forceps to dislocate the lens, then, releasing the grip, expressed the lens with its capsule by pressure from without. Török went a step further, using the forceps to withdraw the lens. Elschnig completed the technique by employing orbital akinesia, steadying the eye by a suture passed round the tendon of the superior rectus muscle, injecting a local anæsthetic substance behind the bulb, suturing the conjunctival wound *et cetera*. Gnanadickam remarks that the retrobulbar injection of a solution of "Novocain" and adrenaline not only completes the anæsthesia of the bulb, but also lowers the intraocular tension

[1] *The Indian Medical Gazette*, October, 1932.

by four to eight millimetres of mercury. After the section and iridectomy, the anterior part of the capsule is grasped by special forceps at a point towards the lower pole of the lens, while gentle counter-pressure is made at the limbus at "six o'clock". The lens is drawn forward while a side-to-side movement is made with the object of rupturing the lower part of the suspensory ligament. As the lower pole of the lens slips forward into the anterior chamber the strabismus hook is made to press gently on its posterior surface, through the cornea. The lens swings forwards and upwards on a hinge consisting of the upper fibres of the suspensory ligament, until its lower pole presents at the wound at the limbus. At this stage the upper fibres of the suspensory ligament are ruptured and the lens, with its capsule, is withdrawn.

Rupture of the capsule is an accident that may occur at any time after it is grasped with the forceps. If the capsule ruptures in front before the lower fibres of the suspensory ligament are broken, the lens may be removed without its capsule. If the posterior part of the capsule ruptures, the whole capsule is pulled away with the forceps and the lens is gently massaged out later. If the capsule ruptures just as the lower fibres of the suspensory ligament are broken, the lens is delivered extracapsularly and the capsule is afterwards drawn away with forceps. The worst accident is rupture of the capsule when the lens presents at the wound; Gnanadickam describes several methods of procedure in this event, the most severe being removal of the whole mass with the vectis, which is employed only as a last resort.

Loss of vitreous humour is apt to occur if the operation does not proceed smoothly, if the eyelids are short and tense, if the patient is very excitable, and so on. Gnanadickam agrees with Smith and Lister that loss of a small amount of vitreous is of no great importance. Gnanadickam points out that some cataracts are not suitable for extraction with forceps. Such, for instance, are the intumescent and Morgagnian cataracts; these are readily removed intracapsularly by the Smith method, but, according to Gnanadickam, they are just as satisfactorily dealt with by extracapsular extraction.

It is impracticable to give a full description of the operation in this place or to discuss fully the various possible complications and difficulties. Gnanadickam's description is clear and full, and illustrated with several excellent, though simple, diagrams. Though he has had experience of the operation for a little longer than two years only, Gnanadickam has performed it on more than one thousand occasions. It is unfortunate that he is unable yet to provide records of the late results of treatment. As far as his experience shows, results have been excellent.

Probably the day will come when the removal of the cataractous lens with its capsule will be the operation of choice. As yet there is no method of intracapsular extraction that can be safely recommended for general use. The Stanculeanu-Tõrõk-Elschnig operation, though not so difficult as Smith's operation, is still very difficult. Every surgeon who has removed a cataract knows how delicate is the lens capsule; it splits almost at the slightest prick; the mere holding of it with forceps must require a delicacy of touch that can be acquired only by long experience. Gnanadickam and others who perform the intracapsular operation regard the loss of vitreous with a complacency that would certainly not be felt by most ophthalmic surgeons. Gnanadickam's paper is a valuable contribution to the study of the treatment of cataract, and as such is worthy of earnest consideration.

CLIMATE AND BLOOD PRESSURE.

THE effects of climate on blood pressure have been studied by numerous investigators. The results have been so variable that they give no real information. It might be expected that hot weather would affect the blood pressure by causing dilatation of the peripheral vessels by decreasing the appetite and increasing hours of rest; some observers believe that the blood pressure is lowered by raised atmospheric temperature, others do not. Hitherto investigators have confined their activities to a study of apparently normal people. Recently I. I. Lemann has recorded the results of his investigations of the influence of a hot climate on persons affected with raised blood pressure.[1] He remarks that, if variations in blood pressure can be caused by variations in climate, they should be much more pronounced when the blood pressure is far above normal. This is not wholly logical; for hyperpiesia depends usually on some pathological condition, which may not be influenced by climatic changes.

Lemann's work was carried out in New Orleans, where the mean monthly maximum temperature varies from 16·9° C. (62·4° F.) in January to 31·8° C. (89·2° F.) in July. The lowest temperature recorded is −9·3° C. (15·3° F.), the highest 39° C. (102·2° F.). "The climate in New Orleans provides an opportunity to study the effect of a long summer with maximum sunlight in contrast to that of a real winter, moderate though it be."

Forty-five patients were studied; their ages ranged from thirty-five to sixty-nine years; eight were males, thirty-seven females. It was found that the blood pressure remained almost constant throughout the year and that any variation that might have occurred from time to time could not be attributable to meteorological conditions. The examination of a small control group over a number of years revealed no pronounced alteration in blood pressure and no evidence of the influence of climate.

The blood pressure varies so greatly as a result of varying physical and mental conditions that an estimation of the effect of meteorological conditions upon it is not easy. Lemann seems to have shown that hot weather *per se* has no influence on abnormally high blood pressure.

[1] *The American Journal of Tropical Medicine*, September, 1932.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Effect of Urine on Hæmolytic Streptococci.

G. H. ROBINSON AND F. A. TAYLOR (*Journal of Infectious Diseases*, March, 1932) have studied the effect of urine on hæmolytic streptococci. They noted that there is a fundamental difference in the food requirements of hæmolytic and non-hæmolytic streptococci, the former requiring a highly complex protein substance, while the latter resemble saprophytes in their ability to thrive on fairly simple chemicals. Normal urine does not contain the substances necessary for the continued multiplication of hæmolytic streptococci. Urea added to broth in amounts considerably less than that found in urine exerts an inhibitory and bactericidal action on the hæmolytic variety. The authors conclude that the frequent occurrence of non-hæmolytic streptococci in urine and the infrequent occurrence of hæmolytic strains are not due to the latter dissociating into the former, but to the inability of the hæmolytic variety to grow in urine. The hæmolytic varieties so rarely recovered probably indicate a rapid excretion from the circulation and not the presence of a hæmolytic streptococcal infection of the urogenital tract.

The Production of Antitoxins in Horses.

G. RAMON AND E. LEMETAYER (*Journal of Immunology*, February, 1932) report successful results in an effort to increase and prolong antitoxin production in injected horses. It had been noted that in any given series of horses injected, one or two of the animals spontaneously produced a marked increase in antitoxic power of serum, the increase being closely related to the occurrence of an abscess at the site of injection of one of the doses of antigen. It was later found that the addition to the antigen of certain substances, such as tapioca or calcium chloride, also increased antitoxin production. The authors selected a number of horses which had been supplying antidiphtheria serum for some time, including certain animals whose antitoxin production had greatly declined. To the three doses of antigen administered during the intervals between monthly bleedings tapioca was added during the first month and calcium chloride during the second, one gramme of either substance being added per one hundred cubic centimetres of antigen. Injections of these mixtures usually produced rather marked local reactions, but after some months the general condition of the animals became good. Determinations of the antitoxin were made each month and it was noted that potency increased. In one animal which had yielded serum for four years, the antitoxin power rose from one hundred and seventy-five to seven hundred units, in another from one hundred and fifty to four hundred units. In recently hyperimmunized horses the rate of increase was also considerable, the titre in one increasing from nine hundred to eighteen hundred units, and in another from three hundred to seven hundred. The inflammatory reaction produced by the injection of the mixture of antigen and tapioca or of antigen and calcium chloride is accompanied by an outpouring of plasma and leucocytes with increased production of tissue cells and other reactions. As a result the antigen becomes enclosed in a pocket or cavity, from which it is slowly absorbed into the circulation.

Blood Cultures in Healthy Persons.

ALLAN R. REITH AND THEODORE L. SQUIER (*Journal of Infectious Diseases*, September-October, 1932) examined blood cultures from two hundred and ninety-three apparently healthy persons. Careful histories and thorough physical examinations, including complete examination of teeth with X rays and examination of sinuses, prostates *et cetera*, were made. The average age of the subjects was thirty-four years. Growth was noted in one hundred and thirteen of the two hundred and ninety-three inoculated media. Of the growths recovered twenty were frank contaminants of the subtilis and sarcina groups, twenty-eight contained staphylococci alone or with frank contaminants, while sixty-five which grew streptococci, diphtheroids, diplococci, *Micrococcus catarrhalis*, colon bacilli or strict anaerobes were regarded as positive. Streptococci were recovered in twenty-four cultures, eight were non-hæmolytic, three slightly hæmolytic, and eight produced typical green colonies. A conspicuous feature of the majority of the diphtheroids recovered was their pronounced pleomorphism. A close relationship of some diphtheroids to streptococci was repeatedly observed. Growths of streptococci or diphtheroids were not usually detected in culture until after seven days' incubation, occasionally not until after fourteen days, and in two instances not until after twenty-one and thirty days respectively. A large majority of the staphylococci were regarded as undoubtedly contaminants. In the group of two hundred and ninety-three persons there were one hundred and ninety-four with chronic focal infection and ninety-nine in whom no focus could be discovered. Twenty-seven *per centum* of cultures from persons with some focal infection were positive, in contrast to 12% from the group in whom no focus could be demonstrated.

Antibody Response to Typhoid Vaccine.

L. TUFT, E. M. YAGLE AND S. ROGERS (*Journal of Infectious Diseases*, February, 1932) report the results of an investigation into the antibody response of mixed typhoid vaccine administered by various routes. Antibody response was measured by complement fixation tests and agglutinating power of the serum. It was noted that in most instances the serum obtained before the administration of vaccine showed no agglutinins and, even when present, they never gave agglutination of typhoid antigen in a dilution of serum of more than one in twenty, of paratyphoid *A* antigen in dilution of more than one in forty (this titre was obtained in three cases), or of paratyphoid *B* antigen in dilution of more than one in eighty (obtained in two instances). In two of the instances named, prophylactic vaccination against typhoid some years before accounted for the presence of the agglutinins noted. Intradermal, subcutaneous, intramuscular and oral methods of administration were investigated. The vaccine used, except in oral administration, consisted of a saline suspension of cresol-killed bacteria containing one billion typhoid organisms and five hundred million each of paratyphoid *A* and *B* per cubic centimetre. Oral administration was obtained by giving three capsules, each containing one billion typhoid organisms, and seven million each of paratyphoid *A* and *B*. The doses administered subcutaneously and intramuscularly were 0·5, 1·0, 1·0 and 1·0 cubic centimetres; intradermal were 0·05, 0·1, 0·15 and 0·2 cubic centimetres. The results showed that local injection produced uniformly better and more persistent response, particularly the intradermal route. It was definitely noted that the older the vaccine used, the less good was the response obtained. Antibody response after oral administration was completely absent. From a therapeutic standpoint the intradermal injection of small doses of a freshly prepared and antigenically potent mixed typhoid vaccine is recommended.

The Isolation of Organisms from Bacillus Proteus.

R. M. FRY (*British Journal of Experimental Pathology*, October, 1932) recommends the following method for the isolation of organisms from *Bacillus proteus*. It is applicable for isolation of any organisms which are not obligate aerobes and the deep colonies of which can be readily identified. The method depends on the fact that in depths of a solid medium *Bacillus proteus* grows in discrete colonies. The material from which a culture is to be made is spread on blood agar, and melted agar at about 45° C. is then poured over the surface of the plate to a depth of two to three millimetres and allowed to set. After incubation it will be found that any colonies of *Bacillus proteus* growing between the two layers of agar show no tendency to spread and colonies of other organisms can be easily picked out from among them. It generally happens that some of the proteus spreads round the edge of the agar on to the upper surface, and to kill these colonies the surface

of the plate is flooded with saturated mercuric chloride solution for about thirty seconds. This is then washed off with tap water and some of the surface growth of proteus is scraped off with the end of a microscope slide to enable the deep colonies to be examined, picked off and subcultured. If the colonies are very crowded, it is advisable to cover the subculture with a layer of agar in the same way and to repeat the whole process. This method is particularly successful in the isolation of hæmolytic streptococci from mixed cultures, as well defined rings of hæmolysis surround the colonies of these organisms in deep cultures. In dealing with anaerobic strains of streptococci it should be remembered that the deep colonies may be very small.

HYGIENE.

Scarlet Fever Control.

FRANK W. LAIDLAW (*New York State Journal of Medicine*, May, 1932) states that scarlet fever control is of little value unless a real investigation is made to find the source of the infection. The source may still be active and other persons infected at the same time. Active search, disregarding crude alibis, will frequently reveal the child with mild unrecognised scarlet fever and permit of its isolation and the quarantining of other contacts.

Infectious Diseases in Schools.

L. R. LEMPRIÈRE (*The Practitioner*, January, 1932) discusses infectious diseases in public schools, their origin, spread and prevention. In 1885 a code of rules for the prevention of infectious diseases in schools was adopted in England; this code has been adhered to for nearly fifty years, with small success. The frequency and extent of infectious diseases in schools has not been lessened, in spite of rigid adherence to these rules, which included the rigid observation of quarantine and segregation of contacts, disinfection of the individual, his clothing, rooms and effects, disinfection of contacts, and spraying of class-rooms and dormitories. Disinfection of the individual, his clothing, and burning of books used is not necessary in measles, rubella, mumps or pertussis; but these measures are necessary in scarlet fever, diphtheria and varicella. Infections as a rule are conveyed from boy to boy; and spread is greater the more the boys are crowded together. Epidemics arise in the first three or four weeks of term from holiday infection; the winter time is the most serious, because the boys tend to crowd together in cinemas, theatres and other closed places during the holidays. Infection during term may be brought in by visitors, tradesmen, by the boys when they are given leave home, or by playing games against infected schools. The extent of an epidemic is impossible to forecast, even when the number of non-immune persons is known. Epidemics vary from year to year. The duration is equally variable. The use of convalescent measles serum is recommended during the incubation period. It has been suggested that contacts should be observed at school and sent home only if they show signs of ill health, in the case of the non-notifiable infections; but it is doubtful if the public would be prepared to accept this system. The present system of sending contacts home is wasteful of time and actually has not decreased the number of those infected in the past.

The Social Burden of the Rheumatic Diseases.

D. M. CONNAN (*Journal of State Medicine*, May, 1932) refers to the Ministry of Health's classification of 1922, set out in Dr. Glover's report on chronic arthritis, Number 52. Sir George Newman has pointed out that acute rheumatism is the cause of heart trouble in 98% of cases in patients under ten years of age. The percentage decreases as the age advances, but even in those under forty years the percentage reaches 60% to 70%. For all practical purposes rheumatism is the cause of deaths from heart disease under the age of forty. The London County Council has a scheme for the supervision and treatment of rheumatism in school children, operated in connexion with the school medical service. At the beginning of 1930, 11,000 names were on the register and 550 were waiting for institutional treatment. In London alone over 2,000 children receive treatment every year for rheumatism and chorea or their sequelæ in the form of rheumatic carditis. Chronic arthritis, including gout, causes only five deaths per thousand. Three times as many deaths occur amongst females as compared with males. Only about three in every thousand patients admitted to large general hospitals are admitted for the treatment of chronic arthritis.

Industrial Rheumatism.

MATTHEW B. RAY (*Journal of State Medicine*, May, 1932) states that age is among the many factors in the causation of rheumatism. True rheumatoid arthritis appears fairly early in life. Infective arthritis as well as fibrositis or muscular rheumatism may arise from early adult age onwards. Gout is a middle age affection, while osteoarthritis is always associated with the degenerative changes of advancing life. True rheumatoid arthritis is much more common in women than in men, and gout is much commoner in men than in women. Osteoarthritis may occur in either sex, affecting the spine and hips in men and the knees in women. The strongest hereditary predisposition is seen in gout, rheumatoid arthritis next, then osteoarthritis. "Diathesis" or predisposition to disease may affect the unified connective tissue system in the embryo, called by Achard "lacunar". Regarding endocrine action, lowered sugar tolerance is highly characteristic of the rheumatic state. Llewellyn points out that not only do goitre and rheumatic disorders arise under the same cosmo-telluric conditions, but the two affections often coexist in the same individual, goitre being usually first to appear. The part played by the skin is important. The regulation of bodily heat is a function of the thyreoid-adrenal-sympathetic system. In long continued cold this system must be adequate. Inadequacy is recognizable in individual types. Such people are happy only in warm climates; in cold climates they are always more or less ailing. They suffer from asthma, constant colds, lumbago, sciatica and the like. Their skins are harsh and dry, and only with difficulty sweat. Along with skin inactivity interference with the flow of blood occurs in the finer capillary beds (Pemberton). Schuloff similarly states that the essential cause of rheumatism is trouble in the capillary circulation of the organs of locomotion. Cold or climatic irritation sets up an autonomic spasm. "The skin is the antennæ of the vegetative nervous system." There is a close connexion between rheumatoid arthritis and anxiety states. A long history of domestic worry often precedes. Nursing sick relatives is apparently a very fruitful cause. Sciatica and brachial neuralgia are very frequently seen in harassed business men who have to carry a heavy load of responsibility. Severe mental shock may immediately precede joint trouble. In investigating factory workers for the pre-rheumatic state, regard should be paid to signs of vasomotor instability, characterized by numbness, tingling, sensation of heat and cold in the hands and arms, experienced long before arthritic signs, and generally in women. Inadequacy of the sympathetic-endocrine system has already been referred to. The designation "frigo-sensitive" is often applied. Previous history of acute or subacute rheumatism or fibrositis in childhood strengthens the opinion.

The Chemical Formula of Vitamin C.

OTTAR RYGH (*Journal of State Medicine*, October, 1932) has determined the chemical formula of vitamin *C*. Narcotine, in which the unripe fruit is rich, is pro-vitamin *C*. As the fruit ripens two orthomethoxy groups lose their methyl group and form an orthodiphenol vitamin *C*. Guinea-pigs are protected by 0·02 to 0·03 milligramme daily. Narcotine is a yellow oil mixed with needle-shaped crystals obtained by concentration by evaporation *in vacuo*. Oil is removed by ether. The clear fluid is rendered faintly alkaline by sodium hydroxide, and narcotine is obtained by evaporating off the ether from the other extraction product of this fluid. All processes must be carried out in the absence of air. Narcotine can be readily synthesized into the orthodiphenol vitamin *C* by heating with strong hydrochloric acid for six days.

Special Articles on Treatment.

(Contributed by request.)

I.

THE TREATMENT OF MIGRAINE.

IF my memory serves me truly, it was of the French physician Labarraque that this story was told, and it was about the time that Dubois Raymond, in Germany, was saying that intracranial vascular spasm explained the paroxysm of migraine, and that Liveing, in London, was having pictures drawn of the characteristic spectral illusions and scintillating scotomata.

Walking one day beyond the walls of Paris, Labarraque happened to experience a severe attack of migraine. The only resting place available was a blacksmith's forge, which he entered and wherein he sat himself down. Explaining his ailment to the smith, he was given this advice: "Eat sparingly of meat, let water be your drink, and take much exercise in the open air."

It is a familiar experience that the vegetarian or lacto-vegetarian diet, forcibly urged by Haig, himself migrainous and a writer upon uricacidæmia, is beneficial. Many a migrainous subject has expressed grateful appreciation of the regimen, and yet it is no panacea; many others have declared it to be void of effect.

Thus early we learn that migraine is a capricious affection. Most of our patients know from sad experience that certain foods produce disastrous effects. Some say that soups, meat and eggs are fatal; others cannot tolerate sugar. Anyhow, it has ever been our maxim to hearken to their caprices, and in doing so we have not known that we were standing on the threshold of a novel form of therapy, one for which we have mainly to thank workers of Continental schools.

Put very briefly and crudely the new assumption is that migraine may be of anaphylactic origin, and so have analogies with asthma and urticaria. This practical observation, among many others, has been made. The ingestion of chocolate in certain subjects has precipitated an attack of migraine and this has been attended by a fall in the leucocyte count. The emerging suggestion is that migraine may be appropriately treated by anti-anaphylactic means. This is the basis of treatment by peptone, either ingested before meals or injected intravenously, of treatment by intramuscular injections of milk and of horse serum, and crotalin (cobra venom).

Personally, I have small experience of such measures, but good results are claimed, and though the exponents admit there is still much to learn, and in particular the biochemical modifications by whose silent and surreptitious agency the migrainous crisis is precipitated, remains to be discovered, the whole subject deserves attention.

To the sufferer from migraine it is always well to preach the importance of a simple life. Abernethy, who humorously remarked that when he had a migraine "he could see no more of his name than the 'knee' (ne) and the 'thigh' (thy)", added: "If you wish to be well you must live on sixpence a day, and earn it yourself."

It is a true observation that he—it is more often a she—who suffers, is a creature of ups and downs, one day depressed, the next full of the *joie de vivre*, and it is during the latter periods that indiscretions are committed which have to be paid for later. As Christiansen, of Copenhagen, said:

> The more regular the life, the more the hours are rigorously divided into periods of work and rest, the more the days are spent in a calm atmosphere free from emotional disturbance and excesses, so much the more will the paroxysms be put off and so much the more, when they do come, will they be tolerable.

Every practitioner recognizes the value of some interesting form of exercise, preferably taken in the open air; he will have notes of some patients whose suffering has not been relieved until they have had long spells at the seashore or in the mountains.

An attack of migraine is often traceable to apparently insignificant cause. "A wheel or a cog out of place throws the whole machinery out of gear." Perhaps the London physician who said that 95% of subjects carried some causal ocular defect, overstepped the mark, but these words of Jelliffe's are something to ponder over:

> It is folly to shut one's eyes to the very evident clinical fact that certain migraines are relieved, if not entirely wiped away, by the correction of some peripheral disorder, sometimes more than one, which has had definite action on the nervous system. Certainly the relief from eyestrain, from diseased turbinates, from adenoids, from constipation, from dysmenorrhœa, from a number of minor yet definite peripheral irritations, will relieve a certain number of patients. In denying any possibility to these influences one errs almost as badly as when maintaining some of them to be the only element in the case, as faddists are doing and always have done.

Turning to well tried methods of treatment by drugs, and thinking not of those persons with simple migraine who scarcely need, and at any rate cannot be bothered with, a prolonged course of medication, but of those unfortunates whose headaches are prostrating, whose general health is impaired by their frequency, and who may be looked upon as examples of "*status migrainicus*", we have these alternatives.

We may follow Sir William Gowers, the first to insist that migraine was a first cousin to epilepsy, and prescribe bromides, and we may prescribe them exactly as we would in a case of epilepsy. One plan is to give a continuous daily dose of approximately forty-five grains; another to divide the periods of treatment into months in this manner, during the first week offer a small daily dose, say, thirty grains, in succeeding weeks, increase the dose by fifteen grains, until in the final week a maximum of seventy-five grains daily is being taken. During the next month work reversely, reducing the daily dose back to thirty grains, and so on for several months.

When bromides fail or disagree, "Luminal" may be resorted to, either as an adjuvant to the bromides or alone. If given alone, it may be spread over a six weeks' course in this manner: three-quarters of a grain thrice a day, twice a day and at bedtime only, spaced over successive periods of a fortnight.

A third drug for continuous use, also dating back to the time of Sir William Gowers, is nitroglycerine, which may be used for several months in doses of one to two grains. Note, however, that administration must be suspended during an attack, because then it will almost assuredly aggravate the headache.

It must be conceded that these forms of treatment often disappoint us; indeed, if we are to be sincere, we must admit that the cure of migraine by drugs is a very uncommon event. At the same time, although we may fail to effect a cure, we may at least mitigate and abbreviate the paroxysms, as well as prolong the intervening periods of freedom.

In the treatment of the attack itself, assuming it to be in its ordinary form, we of course resort to one or a combination of the phalanx of drugs known to form the basis of all "headache cures". To the young woman, however, who carries aspirin tablets in her bag, we offer this advice: "Your tablets are not as innocuous as sugar. If you are to ease your headache, you should take the proper dose and take it in the premonitory stage; also, and this is most important, you will help the drug by sitting or lying down for half an hour, in quiet and under warm covering." Those of the older school are aware that ten grains of Dover's powder is a useful addition to the "headache cure". But the salient point is that none of these very many drugs is for ambulatory use.

The intelligent subject usually knows his own case, whether it is cold to the head or warmth to the feet which gives relief, whether his pillow is better high or low, whether a cup of hot coffee or tea is of use, and so on; hence to advise on these points is like "carrying coals to Newcastle".

As far back as 1765 Robert Whytt said of migraine: "Vomits should be given an hour and a half beforehand." And an emetic or a natural emesis has relieved many a headache. Unfortunately, however, vomiting may persist and abrogate oral administrations. Then we must think of our hypodermic syringe and of what Edinger wrote:

> A word in regard to morphia. I admit freely I cannot get along without this remedy in the treatment of the severest forms. The attack has so extremely depressing an effect upon the psychical condition of the patient, the pains are so great, the general disturbance from vomiting, and, in fact, also from the limited ingestion of food, is so extreme that an injection of morphine is often quite justifiable. Morphinism need never be feared so much as to make us, for this reason, withhold from the patient a remedy of the highest value. The pain and nausea are greatly lessened by the injection of ¼ to ½ a grain.

So many disorders have been ascribed to disease or dysfunction of endocrine glands that it is not surprising to find migraine in this category, and the glands mainly blamed have been the thyreoid and the ovaries, the latter because migraine and the menses so often coincide. Consequently, extracts of these glands have been recommended and favourable results claimed. I must confess to disappointment in my trials of this therapy.

In view of the hypothesis that migraine is a manifestation of intracranial vasomotor spasm, brought about by irritation of the vegetative mechanism (the old view of Dubois Raymond revived), the aid of the surgeon has been invoked and various measures practised to cut off sympathetic control. I have no exprience of cases treated by these measures, but may quote Jefferson:

> Plainly such cases would have to be carefully selected and confined to those who have recognisable vasospasm in superficial vessels, in addition to the conjectural spasm of various arteries within the skull.

Finally, in the stories of Greek medicine, there are instructive references to headaches ameliorated, even cured, by suggestion, or at least by mental influence.

> Agestratos, resorting to the temple at Epidaurus, chanced to fall asleep and dream that Æsculapius came to him and promised him the cure of his headaches, and at the same time gave him valuable hints in wrestling and advised its practice. He woke at daybreak and departed in a state of physical and mental uplift. He practised wrestling assiduously, and not long after was proclaimed a victor at the Nemean games.

A mere suggestion, fortuitously and fortunately received, had changed his mental attitude and provided congenial occupation of mind and body.

And the well known dialogue of Socrates with Charmides, as told by Plato, is worth repeating. Charmides was another victim to headache, and among other things Socrates told him this:

> If the body is to be well you must begin by curing the mind. And the cure, my dear youth, has to be effected by the use of certain charms and these charms are fair words.

To all subjects of migraine, particularly young women in *status migrainicus*, burdened with mournful, even desperate forebodings, be not sparing with "fair words", a "charm" at the disposal of all physicians.

A. W. CAMPBELL, M.D.,

Consulting Neurologist, Royal Alexandra Hospital for Children, The Coast Hospital, Prince of Wales Hospital.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on November 4, 1932, DR. E. S. MEYERS, the President, in the chair.

Anterior Poliomyelitis.

DR. S. F. MCDONALD read a paper entitled: "Signs and Symptoms of Acute Anterior Poliomyelitis in the 1931-1932 Epidemic" (see page 1).

DR. A. E. PATERSON read a paper entitled: "A Few Notes on the Serum Treatment of Acute Anterior Poliomyelitis" (see page 5).

DR. LESLIE GIBSON read a paper entitled: "A Discussion on the Treatment of Anterior Poliomyelitis from the Onset of Paralysis till the End of Spontaneous Recovery of Power in the Muscles" (see page 8).

DR. A. V. MEEHAN read a paper entitled: "Late Treatment of Anterior Poliomyelitis" (see page 10).

DR. J. V. DUHIG showed microscopic slides illustrating the pathology of the condition. He discussed them on the basis of his paper published at page 14.

DR. J. GRAHAME DREW read a paper entitled: "Epidemiology of Anterior Poliomyelitis, with Special Reference to the Queensland Outbreak, 1931-32" (see page 26).

DR. G. A. C. DOUGLAS said his first duty was to express his thanks to the speakers for the large amount of work they had put into the papers, and particularly for the papers as an expression of their personal experiences. Thanks were also due to the Council for arranging such a complete symposium of poliomyelitis. At first it had seemed to be a subject on which enough had been written for the present year and on which there might be nothing fresh to be said. But after hearing the papers Dr. Douglas realized that it was only at the beginning. One could only speak with authority of things which one personally knew, and the papers were valuable from this point. Poliomyelitis was shown to touch every branch of medicine. During an epidemic every febrile condition was poliomyelitis till proved otherwise. Dr. Duhig's slides showed the importance of time, which was the essence of the contract in diagnosis and treatment. The late epidemic was the first in Brisbane in which there had been concerted effort by all branches of the profession, and the results were shown in these papers. The mode of spread of infection was definitely established and the fact that the earlier treatment was given, the more hope there was of success. Serum was useful in the preparalytic stage. In the second stage, where paralysis was established, the question of the value of serum was still an open one. Dr. Duhig's slides explained some apparent discrepancy. The disease started in the neurones and frequently in a few hours the maximum paralysis was attained; in such a case serum would not have much effect. Where paralysis was extending fairly quickly, serum was of use, as generally it stopped the extension of the paralysis. Another point stressed by the papers was that not only must treatment be urgent, but it must be carried on over a long period. It touched the general practitioner, the hospital surgeon, the masseuse, and when it came to non-recoverable cases, the orthopædic surgeon had to make the best of a bad job. One point that required mention was that where an epidemic had occurred in one year, it might occur in the second and third years, and the profession must concentrate on diagnosis, the early use of serum and the effectiveness of serum in stopping or slowing down the paralysis.

DR. HAROLD CRAWFORD joined Dr. Douglas in his congratulations. Dr. McDonald had shown that there were three early stages—the initial, the latent and the preparalytic. Dr. Crawford thought that a lumbar puncture should not be done before the preparalytic stage, as previous to this there was no change in the cerebro-spinal fluid. He did not agree that a lumbar puncture was only a minor operation, and thought that it should not be done, or serum given in the absence of the cardinal signs of stiffness of the spine *et cetera*. He agreed with Dr. Paterson on the point of giving serum in cases in which paralysis had already occurred; Amoss said the same. Dr. Gibson had brought out the absolute necessity for splinting the trunk muscles; in the previous epidemics all attention had been paid to the extremities, and cases of scoliosis were now being seen as the result of the previous neglect. One could infer from this that all

children with poliomyelitis should be treated in institutions. No kneading should be done to the muscles at any stage of the paralysis, but only gentle massage. Dr. Crawford considered that a number of the febrile attacks occurring in children during an epidemic were really abortive attacks of poliomyelitis, and that they conferred an active immunity. Dr. Duhig had shown that up to the first year the incidence of poliomyelitis was very rare. This was probably due to inherited immunity. Dr. Crawford quoted the neutralization tests by Kramer, who showed by such tests that immunity could be obtained by those in close contact with a patient, without such persons developing the disease. When tested within a few days, they were non-immune, but were immune when retested in five months from the date of contact. Would pooled adult serum be satisfactory if the hospital ran out of serum? Dr. Meehan showed that the care of poliomyelitis after two years was a case for the specialist.

DR. T. HENRY asked had many cases been seen in the recent epidemic, which appeared to be purely encephalitic, showing no paralysis, but with a febrile condition and definite signs of irritation.

DR. W. J. SAXTON thanked the speakers for their papers. He was interested to hear Dr. Duhig's remarks on the destruction of the nerve cells by the virus, as he had thought the condition was due to nutritional disturbance of the cells. He did not agree with Dr. Crawford that lumbar puncture was not a minor operation in the hands of any person used to it, and he considered that if a lumbar puncture were done, serum should be at hand to be given immediately.

DR. G. A. McLEAN thanked the speakers for their illuminating papers. He referred to the question of early diagnosis, as relating to the general practitioner. Early diagnosis could be difficult, as shown in the recent epidemic. Possibly one developed an intuition in these cases; but if one did not, it was better to do a lumbar puncture to aid diagnosis and to have serum given early. Some cases developed quickly and some slowly; he considered it was too late to wait for spinal rigidity; one had to consider each case as a fulminating one and treat it accordingly. The question of isolation arose, whom to isolate and how strictly. If the disease were communicated by droplet infection, should there not be strict isolation of all relatives, which would not be popular, particularly with the working people.

DR. A. J. CAMERON said Dr. Drew had mentioned communication by rail as a factor in the spread of the disease. Dr. Cameron pointed out that there had been one case from Booval, one from Rosewood and none from Ipswich, which was connected by rail to these places and to Brisbane, being only twenty-five miles from the latter.

DR. J. A. GOLDSMID said Dr. Duhig had pointed out that the virus ascended along the neurones. If such were the case, what was the explanation of the benefit derived from intrathecal and intravenous injections, and what objection was there to using purely intramuscular injections?

Dr. McDonald, in reply to Dr. Crawford, said that he considered lumbar puncture justifiable at the earliest sign of spinal rigidity in the preparalytic stage. Possibly immunity was due to various almost febrile conditions, quite disregarded, which were really abortive attacks. As regards the potency of the local serum, he again instanced Dr. Masel's case. Two children at present in the Hospital for Sick Children had been treated with pooled adult serum; their paralyses were extensive.

In reply to Dr. Henry he stated that in all he had seen six cases of the encephalitic type. All had been fatal except one (Chart V).

With Dr. Saxton and Dr. McLean he was in complete accord as regards the importance of early diagnosis and serum administration. Puncture should be performed if necessary on suspicion, but if puncture was to be done, everything should be at hand — microscope, counting apparatus and serum, so that no time need be wasted. To do as not infrequently was done, to do a puncture, to bring fluid in five miles for examination and to return with serum meant delay and disturbance of cerebro-spinal balance, so that rapid extensions might occur. One sad experience of his own had convinced him of the importance of Dr. McNamara's views in this respect. As to the route of serum administration, he would leave Dr. Duhig to answer Dr. Goldsmid.

Dr. Duhig, in reply to Dr. Crawford, said that undoubtedly pooled adult serum could be used. A large majority of the people in the population were exposed to poliomyelitis and must surely become immune. Rhoads, in the course of testing the immunity of sucklings, tested twelve mothers and new-born infants, and found that 83% were immune. So the large majority of adults in any population must be immune. The potency of so-called normal serum was found by Park to be only about half that of a person with residual paralysis. Normal serum should not be used intrathecally, as it might cause serious disturbance; if only normal serum were available, it should be given intramuscularly. Howitt, in America, had prepared sheep and goat serum with excellent results, when it was given intramuscularly. In the 1928 epidemic in Manitoba the intramuscular method had been used almost exclusively; the infection was a virulent one, and 93% of patients had recovered at a stage where normally convalescent serum would be given. Dr. Macnamara recommended intrathecal use of serum, but Dr. Duhig thought that the patient should be brought under the influence of the special therapy as soon as possible and that the intravenous method was the best. The patient should be kept under its influence by intramuscular injections.

With regard to the pathogenesis of the disease he himself had had no experimental knowledge of it at all. Slides showed that there could be widespread interference with the nutrition of the cells. The views he had expressed were those of Hurst, who was one of the best authorities in the world. There must be a decussating mechanism involved, as shown by his experiments with intranasal and sciatic nerve injections. Two workers in America had divided the spinal cord and shown the virus in the upper part of the cord and none in the lower part. The spread was almost always axonal.

Dr. Paterson, in reply, said he thought the diagnosis of the disease was difficult. One could generally get a fair idea as to the spinal rigidity, but before this one could not say whether the child was suffering from poliomyelitis. He himself treated a patient as suffering from poliomyelitis until proof one way or the other could be made by lumbar puncture. Even in doubtful cases the patient had been kept in hospital for twenty-four or forty-eight hours.

With regard to the administration of serum, he had only his experience to go on. Judging from the charts, those patients who had made the quickest recovery were the ones that had had intravenous and intrathecal serum together. They had a profound reaction and were very ill, but recovered. At times the stock of serum had been very low in Brisbane—it had once been down to ten cubic centimetres—and patients with mild infections had received only either intrathecal or intravenous injections of serum. Some of the patients whose infections had seemed mild and who had had a low cell count, were still in the hospital.

In reply to Dr. Henry, Dr. Paterson said he had seen the encephalitic type of case; these had all had a low cell count.

Dr. Gibson said that muscular pain was definite, but passed off quickly, generally after three weeks. Pain and stiffness in the joints sometimes appeared a few months after the onset. It was certain to occur in a slight degree after use of plaster and immobilization, but was not permanent, and full movement could be obtained after treatment by a masseuse.

Correspondence.

A MEDICAL HANDBOOK.

SIR: In your issue of September 17, 1932, there is, under the guise of a review of one of my recent books, a frankly

hostile attitude taken towards me. You will therefore, I am sure, allow me space to reply.

I do not complain that the review of my book is hostile, although I may say that your review is the sole jarring note in a whole chorus of eulogy towards the book in English and American medical journals.

But what I do complain about is that your review is not informative and that your reviewer misses the whole point of my book because he has not troubled to read it carefully. The review is cynical, slapdash and sarcastic, and in three places, where alleged quotations are given, it is grossly inaccurate. In other places it is grossly unfair in the way quotations from the book are stripped from their context.

I know quite well that an anonymous reviewer has no soul to be damned, but as his identity is known to me, I should like it to be publicly known that he will find he has a body to be kicked if ever I find myself within a hundred miles of him.

Yours, etc.,

G. F. WALKER, M.D., M.R.C.P. (London).

Sunderland,
England,
November 12, 1932.

MODERN OPTOMETRY.

SIR: I have read with interest your editorial of December 10, 1932, on "Modern Optometry", and I appreciate the effort made to warn the public and general practitioners of the dangers of optometry when the optometrist claims to be able to differentiate between the states of health and disease in the human eye; not to mention when he studies such subjects as "blood pressure", "muscular anomalies", "dental sepsis" and "pathology", and receives lectures in such subjects by men who make no claim to having any medical qualifications whatsoever. The matter would be ludicrous were it not so tragic from the point of view of the public.

I daily hear the cry from patients in distress: "Oh, I wish I had known in time, doctor; but I thought I only needed glasses, and that if I went to an optician he would be capable of prescribing suitable glasses, and would be capable of telling me if there was anything else the matter with my eyes."

This is often followed by an accusation levelled at the medical profession generally for not carrying out the ideals of the profession sufficiently to make some effort to educate the public in such matters by medium of the public Press.

My reply is that for obvious ethical reasons individuals cannot warn the public (through the public Press), that the public Press has had many opportunities of publishing reprints from such journals as yours and *The British Medical Journal*, which have published on various occasions very powerful editorials covering the whole range of the menace of the optometrist to the community.

When one stops to think what must be the value of the advertisements of spectacle makers in the public Press, one can realize that it would indeed be a public spirited paper which would reprint for the benefit of the public such editorials as have appeared in the past in *The British Medical Journal* compiled by the men most competent in the British Empire to express such opinions.

From time to time we see political activities with regard to various bills for the registration of sight testers, and for the granting of various powers to them under licence; but, as a minister of a certain government once said to me after an interview with the representatives of a medical association of high standing with regard to a projected bill: "Such bodies cannot expect us to take serious note of their representations because they are non-political, and do not subscribe to party funds." I feel sure such business interests would always tend to negative the efforts of the profession generally to give ample warnings to the public of the dangers which they run, and it is only such papers as your excellent journal that are in a position to carry forward the work of public education by adding to the knowledge of the subject available to the general physician and surgeon.

Your remarks *re* the value of early diagnosis in the treatment of disease, both ocular disease and general constitutional disturbances (which first manifest themselves to the patient as some visual anomaly which the patient in almost 100% of cases personally attributes to the need of glasses), are appreciated thoroughly.

Your remark that there is a place in the community for the optometrist is also appreciated, but is it made clear what that place is?

There is an increasing need for competent spectacle makers. The modern tendency is to mount factory made lenses in standard ready-made frames, and call them spectacles or glasses. The difference between these and spectacles made by a craftsman is the difference between a shop-made boiler suit and a suit of clothes made by a high-class tailor.

However, the need for a refractionist without medical qualifications, though admitted, is daily becoming less for the following reasons. Firstly, though still quite inadequate, the supply of trained ophthalmologists is rapidly increasing. Today in New South Wales the number of graduates of medicine practising ophthalmology alone is approaching the century, in addition to many doing refractions in the course of their general practices. I venture to forecast that within the next decade this number will be at least quadrupled, and this because the public, in spite of the attitude of the public Press, is slowly but surely becoming educated to desire and demand the services of the trained ophthalmologist for prescribing its glasses.

Already, in addition to forty odd ophthalmologists in Macquarie Street, almost all country towns of any size have one or more. In the country particularly, where the ailments of the populace and their treatment are probably more freely discussed amongst themselves than in the city, one is struck by the rapid disappearance of the hawking optometrist who such a very short time ago was so ubiquitous.

There is one phrase in your editorial, Sir, to which I must take exception, though I think the meaning of the written word was probably not intended, that is: "Although some optometrists are expert in the estimation of a refraction and can correct it by ordering suitable spectacles."

Now, while quite willing to admit that in a small percentage of cases it is possible to refract accurately without the use of mydriatics or cycoplegics by the use of various machines, and by subjective tests such as "fogging tests" of various kinds, I would point out that the great and universal stumbling block to refractions without the use of drugs is that the eye is not a mechanical camera, but a mass of living tissue, not only susceptible to influence by all systemic derangements, but capable of adjustments of its refraction by its own inherent muscular powers, not only in accommodation, but in ordinary distant vision. This muscular adjustment is involuntary and automatic, and the muscles responsible vary in their powers in different individuals, in different states of health, and at different ages, and frequently develop spasmodic contractions.

The cases under forty years of age capable of certain accurate refraction without cycoplegics are few and far between; the cases capable of such refraction in childhood are practically non-existent. After forty years of age the refraction of the eye is so bound up with its reaction to pathological conditions that a thorough examination by a man qualified to understand the physiology and pathology of the whole human body and its relation to a refractive error is essential for safety.

In conclusion, these are days of progress. The spectacle maker must feel that there is becoming less and less demand for his services as a refractionist, as the better educated public demands more and more the services of medically trained ophthalmologists.

This has been evidenced in the past by frantic efforts of the spectacle making trade to have some form of registration, which naturally limits the numbers practising that trade. In spite of this, public education and enlightenment progress, and we now have frantic efforts by the optometric trade to invade the realm of medicine further by the investigation of such subjects as "blood pressure", "dental sepsis", *et cetera*.

However, fate is inexorable; the refracting optometrist is, in my opinion, doomed; not through lack of political recognition and activities, but due to the enlightenment of the general public. He will be entirely replaced by the medically trained ophthalmologist as inevitably as the apothecary was replaced by the modern physician and the barber by the modern surgeon.

In so far as the medical profession is concerned, it behoves us to supply the demand for trained medical ophthalmologists, and it behoves the medical educationalists to provide increased facilities for ophthalmological teaching, as the necessity for such increased facilities occurs.

Once more, Sir, I appreciate your editorial. My excuse for this letter is that I hope it will emphasize the points which you make.

Yours, etc.,

F. GUY ANTILL POCKLEY.

223, Macquarie Street, Sydney,
December 13, 1932.

SIR: Apparently your leader of the tenth instant has served one very useful function in that it has provoked a reply from the Secretary of the Optometrists' Institute, who now officially states in the public Press what the aims and claims of this body really are (*vide Sun*, December 14, 1932).

It seems that what mainly arouses his ire is your remark: "What is needed is a realization by the public that only medically trained persons are competent to diagnose disease processes", as he writes: "It is a gross misstatement to suggest that the qualified optometrist cannot diagnose either the presence or nature of ophthalmic disease." The medical specialist, experienced in the pitfalls and difficulties of diagnosis, may be pardoned for doubting whether, in the light of his experience, the Secretary's "*ipse dixit*" settles the question. On reading this outburst, one might at first be inclined to treat the letter as a joke were it not for the fact that being official one must credit these gentlemen with really believing themselves capable and competent to do what their mouthpiece says they can. To my mind, in this lies the real danger to the public, for, to again borrow from your leader: "To deal with arrogance such as that shown by these modern optometrists is a difficult matter."

Apart from private practice, any specialist with hospital experience cannot fail at times to be struck by the utter incompetence and callous disregard for the patient's welfare that is shown by some of these modern optometrists. On the one hand one is consulted by those whose refraction has been wrongly corrected, on the other by those unfortunates who have been given the assurance that their eyes are perfectly healthy and that all their ills will be cured by a pair of glasses, but who finally are compelled to bring their troubles to the hospital, which usually prove to be due to undiagnosed disease processes and then generally when the proper time for effective treatment has long passed. The former as a rule suffer no more disability than an aggravation of their original constant headaches and nervous irritability, but what of the latter—those helpless unfortunates who have been lulled into a sense of false security by these gentry, who have shown themselves possessed of even less than the proverbial little knowledge that is dangerous?

Yours, etc.,

F. ABBEY WIESENER.

"Harley",
143, Macquarie Street,
Sydney,
December 19, 1932.

SIR: Our attention has been drawn to the article that appears in your journal of December 10, 1932.

We would like to clearly point out that the Institute referred to does not represent the views of the whole of the registered optometrists and opticians of this State.

You also mention that optometry is a skilled occupation and some optometrists are highly skilled in their work, to which we fully subscribe.

At a recent lecture before the members of the Opticians and Optometrists Association of New South Wales, it was pointed out that no optometrist had the right to trespass upon the medical field, advise or express opinions outside of refraction of the eye. We do not believe, nor is it the practice of our members, to make any attempt to diagnose diseases of the eye.

It is our policy and our practice to advise our clients to consult an oculist if any abnormal condition is observed.

We do believe, however, in a community like ours, that there is a place for the legitimate optometrist, who must exist and must be considered as a help and an asset to the public.

The legitimate optometrist has also been the medium in sending along to the doctors many cases that would never have got there, or they would probably have gone too late, if it were not for his timely advice.

The feeling of this Association, representing over two hundred members of the optometrical and optical profession, is that the curriculum as set by the Optometrical Board is not necessary, and the educational needs for a registered optometrist are only to have sufficient knowledge, not to be a danger to the public.

Trusting you will afford us space in your valuable journal for this reply to the above mentioned article.

Yours, etc.,

JOHN D. NOTT,
Honorary Secretary,
The Opticians and Optometrists Association of New South Wales.

Challis House,
4, Martin Place,
Sydney,
December 21, 1932.

PROSTATECTOMY.

SIR: I regret that Dr. Keith Kirkland was not more explicit in his letter of December 10, 1932, when he said: "It is not so reassuring to inspect the post-operative condition of inmates of the State Hospital at Lidcombe".

He meant, no doubt, to infer that the post-operative condition of patients transferred to Lidcombe State Hospital after operation at other hospitals was not reassuring.

He refers to a state of affairs dating back some five or six years.

None of these cases of suprapubic sinus had been subjected to the Harris operation.

I understand that this matter will be the subject of a communication from our Honorary Surgeon, Dr. Darling.

Yours, etc.,

H. BARET,
Superintendent.

Lidcombe State Hospital and Home,
Lidcombe, New South Wales,
December 14, 1932.

A READERSHIP IN PHYSIOLOGY.

SIR: I am directed by the Prime Minister to inform you that the University of London advises that there is a vacancy for a Readership in Physiology at Guy's Hospital Medical School at a salary of £800 *per annum*; the clinical side of physiology is stressed. Application should be made before February 17 to the University of London.

It is suggested that you might give such publicity as you can to this opportunity for medical graduates to carry out post-graduate work in England.

Yours, etc.,

J. H. L. CUMPSTON, M.D.,
Director-General of Health.

Canberra, F.C.T.
December 16, 1932.

THE FUTURE OF MEDICINE AS A PROFESSION.

SIR: Your correspondents have asked for some lead in efforts to improve the prospects of medicine as a career and livelihood. The first essential is to recognize the evils requiring correction: (i) The inroad of the public hospital into private practice, (ii) overcrowding of the profession, (iii) overlapping of general and consulting work.

The public hospitals are attracting and admitting, especially to the specialist departments, far too many patients well able to pay some private consulting fee, for the reasons that they offer a service greatly in excess of that to be obtained in the majority of private institutions and that the patient is easily able to prove at the moment that he is without visible means, as he always will do until we are able to reach him before the movies, the hotels, the races and such like have had their pickings of his income; or until we find a means of proving him to be *minus* the difference between the hospital's five shillings and the private doctor's ten shillings and sixpence.

The remedy lies in the following:

(*a*) Not as Dr. Woodburn suggests, to lower the standard of comfort of the public hospital, but to raise the standard of attractiveness and equipment of the private institutions; a result to be achieved only by the activity of the medical men themselves, aided perhaps by the State in an effort to develop forthwith, larger and modern, private and intermediate units in various districts and towns. The public hospitals are likely for a long time to shelve the question of the intermediate and private bed on account of shortage of funds and space while the benefits of such a development, when it does come, are likely to be for the members of the honorary staff rather than for the profession as a whole. I suggest that doctors and matrons should recognize this as their problem for their own solution, and get together in appropriate localities and organize their own community unit by a process of cooperative subscription of capital and resources, absorbing and eliminating the smaller and poorly equipped nursing homes so that there will result an institution which will offer the same if not better X ray, pathological, and general or special services as the existing but superior public institution.

(*b*) That admission to the wards and out-patient departments should be granted only on the production of an outside doctor's certificate, which should be on a standard form which should include a statutory declaration of the patient's means. The effect would be that all sections of the community would be obliged either to make some provision for sickness by joining a lodge, or must be prepared to pay at the time of illness, the State meeting the subscriptions of the truly destitute after investigation as rigid as the present dole system now is. Here is no demand for the withdrawal of our services so long given to sweet charity; the consultant and the major hospital services would remain freely given to those recommended as in such need. On the other hand, the State and the habitual sponger would find themselves performing a duty long overdue to our profession. Overcrowding of the profession can be remedied reasonably, only by a bold dissemination of the facts from time to time, reaching right into the school and homes of prospective matriculants and freshmen undergraduates, and at the same time endeavouring to be constructive by cooperating with our universities in developing new faculties which will provide some alternative to satisfy the ambitions of young men and women wishing to follow a professional career.

(*c*) Could our profession better organize itself on the lines suggested, with fewer members and more paying patients, there then appears to remain the fault within our own ranks of unrestricted competition between the general practitioner and the specialist or consultant, a fault itself the result of the existing conditions, but tending to perpetuate the state of uncertainty of income. The development of privately controlled community units will create a demand for specialists decentralized from the large cities. The general practitioner must recognize that these men make possible a system which directs patients from the free hospital to the community unit where he is able to keep them under his own care. The specialist must in turn realize that the treatment of general types of cases is not within his sphere. Such an attitude will make possible the development of consulting units in the larger outlying centres, with the result that a check will be placed on the flow of patients to the big city centres where more than half turn their thoughts to the public hospital as the natural place to seek specialist advice and treatment. I do not attempt to be able to suggest a remedy for all our troubles, but I do feel that of our own indulgence the faults have arisen, and only by our own efforts will a correction be achieved; we are well organized and powerful in our British Medical Association, but medical politics has not been a prominent feature in our activities. I now suggest the formation of a section of medical politics within the British Medical Association whose duty it shall be to constantly keep abreast of problems such as this; a general meeting early in the new year could set the ball rolling.

Yours, etc.,

DOUGLAS G. CARRUTHERS.

183, Macquarie Street, Sydney,
December 23, 1932.

SIR: I would like to add a few words to the suggestions put forward by Dr. J. Woodburn in his letter on "The Future of Medicine as a Profession", published in today's issue of THE MEDICAL JOURNAL OF AUSTRALIA. I heartily agree with the sentiments and suggestions which he has put forward.

It is amazing when one casually counts the number of hospitals situated within the boundaries of Sydney and its suburbs where treatment of all kinds of cases, both medical, surgical, gynæcological, and others requiring special investigations, can be obtained free. It is still more striking when one considers that the population in this area is comparatively little more than one million.

I think that a great deal can be learnt from the hospitals overseas—especially Scotland—for human psychology is brought to a fine art there. The free hospitals in Edinburgh and Glasgow, and the new Royal Northern Infirmary at Inverness, are all run on strict institutional lines embodying some of the suggestions which Dr. Woodburn mentions in his letter, but, in addition to the insistence upon uniformity of dress of the in-patients *et cetera*, it is stated openly—usually over the entrance gates or hall—that "the institution is dedicated for the treatment of the sick poor".

This simple fact touches personal pride, and a surprising number of patients who are able to afford only a small to moderate amount, prefer to enter the intermediate hospitals or wards rather than allow their friends to visit them or know that they are receiving medical attention and treatment at institutions which are dedicated to the poor. Observance of this psychological fact would probably greatly assist the future of medicine as a profession.

Yours, etc.,

D. G. MAITLAND, M.B., B.S., B.Sc.

B.M.A. House,
137, Macquarie Street, Sydney,
December 24, 1932.

New Year Honours.

AMONG those upon whom His Majesty the King has been pleased to confer honours are two medical practitioners.

Sir Thomas Horder, Physician-in-Ordinary to the Prince of Wales, has been elevated to the peerage.

Dr. John Lawrance McKelvey, of Sydney, New South Wales, has received the honour of knighthood.

The congratulations of the medical profession are extended to Lord Horder and to Sir John McKelvey.

Books Received.

AN INTRODUCTION TO SEXUAL HYGIENE, by A. Buschke, M.D., and F. Jacobsohn, M.D., translated by E. and C. Paul; 1932. London: George Routledge and Sons, Limited; Australia: Angus and Robertson. Demy 8vo., pp. 201, with illustrations. Price: 11s. net.

TALENTS AND TEMPERAMENTS: THE PSYCHOLOGY OF VOCATIONAL GUIDANCE, by A. Macrae, M.A., M.B.; 1932. London: Nisbet and Company, Limited. Crown 8vo., pp. 220. Price: 5s. net.

SOUND CONDUCTION AND HEARING: AN HISTORICAL, CRITICAL AND EXPERIMENTAL STUDY, by A. Zünd-Burguet, translated by M. Yearsley, F.R.C.S.; 1932. London: John Bale, Sons and Danielsson, Limited. Demy 8vo., pp. 139. Price: 8s. net.

CORRECTION OF DEFECTIVE SPEECH: A COMPLETE MANUAL OF PSYCHO-PHYSIOLOGICAL TECHNIQUE FOR THE TREATMENT AND CORRECTION OF THE DEFECTS OF SPEECH, by E. B. Twitmyer, Ph.D., and Y. S. Nathanson, Ph.D.; 1932. Philadelphia: P. Blakiston's Son and Company. Demy 8vo., pp. 428.

WHAT BUTTERFLY IS THAT? A GUIDE TO THE BUTTERFLIES OF AUSTRALIA, by G. A. Waterhouse, D.Sc., B.E., F.E.S., F.R.Z.S., with illustrations by N. W. Cayley; 1932. Australia: Angus and Robertson. Royal 8vo., pp. 291, with illustrations. Price: 12s. 6d. net.

ANATOMY, DESCRIPTIVE AND APPLIED, by Henry Gray, F.R.S., F.R.C.S.; Twenty-Fifth Edition; Edited by T. B. Johnston, M.B., Ch.B.; 1932. London: Longmans, Green and Company. Royal 8vo., pp. 1492, with 1303 illustrations, of which 607 are coloured. Price: 42s. net.

MONOGRAPHS OF THE BAKER INSTITUTE OF MEDICAL RESEARCH: Number I: PRACTICAL ANÆSTHESIA, by the Anæsthetic Staff of the Alfred Hospital, Melbourne; 1932. Australia: Australasian Medical Publishing Company, Limited. Demy 8vo., pp. 249, with illustrations. Price: 10s. 6d. net.

THE INJECTION TREATMENT OF VARICOSE VEINS, HÆMORRHOIDS AND OTHER CONDITIONS, by R. H. Maingot, F.R.C.S.E.; 1932. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 112. Price: 4s. net.

TUBERCULOSIS OF THE LUNGS: A SEMI-POPULAR STUDY OF THE DISEASE AND ITS TREATMENT, by S. L. Piplani, B.Sc., with the assistance of J. N. Piplani, M.B., B.S., D.O.M.S.; 1932. London: John Bale, Sons and Danielsson, Limited. Demy 8vo., pp. 205, with illustrations. Price: 15s. net.

LECTURES ON MIDWIFERY AND INFANT CARE: A NEW ZEALAND COURSE, by T. F. Corkill, M.D., M.R.C.P.; 1932. New Zealand: Coulls Somerville Wilkie, Limited. Demy 8vo., pp. 419. Price: 12s. 6d. net.

THE HOSPITAL ALMONER: A BRIEF STUDY OF HOSPITAL SOCIAL SERVICE IN GREAT BRITAIN; 1932. London: The Hospital Almoners' Association. Demy 8vo., pp. 31.

Medical Appointments.

Dr. E. H. Barrett has been appointed a Resident Medical Officer at the Adelaide Hospital, South Australia.

* * *

Dr. R. B. Wade (B.M.A.) has been appointed President and Dr. A. A. Palmer (B.M.A.) Vice-President of the Board of Management of the Royal Alexandra Hospital for Children, Sydney.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought etc., see "Advertiser", pages xvi, xvii and xviii.

BRISBANE MATER CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: House Physician, House Surgeon.

DIRECTOR-GENERAL OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Honorary Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

NEW SOUTH WALES COMMUNITY HOSPITAL, SYDNEY, NEW SOUTH WALES: Staff Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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